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# Differential Susceptibility to Social Status

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# Differential Susceptibility to Social Status

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# Differential Susceptibility to Social Status

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The diathesis-stress model focuses on the interaction between gene polymorphisms and *negative* environmental conditions (i.e., stressors); however, Belsky and Pluess (2009) recently proposed an alternative to diathesis-stress: the differential susceptibility hypothesis, which states that some individuals may be predisposed to be more adversely affected by negative environments but, also, to benefit more from positive environments. Nevertheless, the differential susceptibly hypothesis has not been rigorous tested. Thus, the purpose of this study was to test the differential susceptibility hypothesis by examining individual differences in men's testosterone, behavioral, and psychological responses to social status as a function of the serotonin transporter promoter region polymorphism (5-HTTLPR), which was cited by Belsky and Pluess as a potential "plasticity gene" because one variant – the long (*l*) allele – appears to be associated with lower susceptibility/plasticity and another – the short allele (*s*) – appears to be associated with higher susceptibility/plasticity.

In this study, groups of 3-4 male participants were allowed to socialize before being told that they were part of a larger initiative to create a student-run Honor Committee. They were asked to nominate one person to be the leader and one person to not be on the committee. Then, participants were told privately that everyone voted them to either (1) be the leader or (2) not be on the committee. Salivary hormone samples

were collected at baseline and 20 minutes after vote feedback. In addition, after receiving the vote feedback, participants completed a series of dating anxiety and mate preference tasks and were given the option to examine an "actual honor violation" case either alone or as part of the committee.

The results support the differential susceptibility hypothesis. In terms of testosterone response, *ss* individuals showed both greater reactivity and differential responses to vote feedback. Furthermore, the testosterone responses of *ss* individuals were moderated by basal cortisol, which is associated with approach/avoidance behavior (Kagan et al., 2003; Shoal, Giancola, & Kirillova, 2003). In addition, *ss* individuals' decisions to work on the committee or work alone and responses to the mating tasks were dependent upon the vote feedback, whereas *l*-carriers' decisions and responses were not.

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# Chapter I: Introduction

## GENE-ENVIRONMENT INTERACTIONS

Many individual differences may be best described as products of gene-environment interactions. One of the most influential paradigms of gene-environment interaction is the diathesis-stress model, which states that individuals who carry certain "vulnerability genes" (or heritable risk factors) are at an increased risk of suffering health problems and developing psychiatric disorders under negative or adverse environmental conditions (e.g., stress). Nevertheless, from an evolutionary perspective, the diathesis-stress model is problematic. If individuals with a given gene variant (or allele) performed worse under stress but performed no better under non-stress conditions than individuals without it, natural selection would have eliminated that allele from the population (Homberg & Lesch, 2011). The persistence of so-called "vulnerability genes" in the population, however, suggests that they must have conferred some adaptive benefit(s). Unfortunately, because the diathesis-stress model only addresses how individuals respond to adversity, researchers have either failed to consider or ignored data concerning how individuals respond to conditions other than adversity, in which such advantages might become apparent (Belsky et al., 2009). Thus, our understanding of gene-environment interactions, particularly in the case of "vulnerability genes," would likely benefit from a more evolutionary approach.

Of course, explaining individual differences from an evolutionary perspective is also a relatively new endeavor, since evolutionary psychologists' primary objective has been to test hypotheses pertaining to species-typical ("universal") and sex-typical psychological adaptations (Buss, 2009). Some evolutionary psychologists even view individual differences in humans as the result of random genetic mutations maintained in

the population either because they result in phenotypic differences that do not affect the function and fitness consequences of adaptive mechanisms (Tooby & Cosmides, 1990) or because natural selection cannot out-pace our relatively high mutation rates (Keller & Miller, 2006). Nevertheless, others argue that some individual differences reflect evolved psychological mechanisms (e.g., Nettle, 2006). They note that differences in traits such as temperament and personality cannot be explained as random genetic mutations because random mutations tend to be rare, have mild yet harmful phenotypic effects, and are ultimately selected out of the population (Penke, Denissen, & Miller, 2007, p. 561). On the contrary, not only are many individual differences in humans highly heritable (Plomin, DeFries, McClearn, & Rutter, 2008), but they have also been linked to specific polymorphisms with prevalence rates significantly greater than 1% (Ebstein, 2006; Kidd, 2006). Furthermore, just as evolutionary biologists studying individual differences in animals have observed (e.g., Dall, Houston, & McNamara, 2004; Wilson, 1998; Wolf, van Doorn, Leimer, & Weissing, 2007), evolutionary psychologists are finding that individual differences in humans do have significant effects on fitness-related outcomes and, thus, likely serve (or served) adaptive functions (e.g., Buss & Greiling, 1999; Eaves, Martin, Heath, Hewitt, & Neale, 1990; Friedman et al., 1995; Nettle, 2005; Ozer & Benet-Martinez, 2006).

Still, although evolutionary psychologists are beginning to view individual differences as adaptive, understanding the role of gene-environment interactions in the development of individual differences from an evolutionary perspective has also been met with debate about whether those differences reflect phenotypic plasticity or alternative phenotypes. Phenotypic plasticity is the ability of a genotype to produce different phenotypes in response to the environment. This implies a universal psychological architecture and emphasizes the role of the environment in shaping

individual differences (Shackelford, 2006; Troisi, 2005). For instance, exposure to high levels of stress during early childhood may alter the expression of the genes responsible for the stress response system, resulting in a highly reactive phenotype and, ultimately, an adult who is more sensitive to stress (Boyce & Ellis, 2005; Ellis & Boyce, 2008; Ellis, Essex, & Boyce, 2005; Ellis, Jackson, & Boyce, 2006). However, phenotypic plasticity does not explain the genetic variation underlying individual differences. Moreover, while it may seem that a plastic phenotype would be more efficient and advantageous than a less plastic phenotype, it is not without its costs and limitations, such as the energetic costs of maintaining more reactive regulatory mechanisms and the risk of developmental instability (see DeWitt, Sih, & Sloan, 1998; Relyea, 2002).

Thus, individual differences may instead reflect alternative phenotypes, in which case different genotypes produce different phenotypes that solve the same adaptive problem (e.g., reproductive success is achieved through either high mating effort or high parenting effort, Rowe, Varsonyi, & Figueredo, 1998). Each phenotype fairs better than the others under certain environmental conditions, but they are maintained in equilibrium in the population because their fitness outcomes are equal when averaged across environments (see Penke et al., 2007). However, this perspective does not address the fact that individuals exhibit considerable behavioral variability; and neither perspective really addresses the fact that there are also individual differences in within-person variability (Fleeson, 2001).

## DIFFERENTIAL SUSCEPTIBILITY

Recently, using evolutionary principles, Belsky and Pluess (2009) developed the differential susceptibility hypothesis to explain individual differences in susceptibility to environmental factors. In essence, they extend diathesis-stress by arguing that some

individuals may be predisposed to be more reactive, in general, by virtue of genetic differences in their levels of sensory sensitivity and physiological reactivity. One advantage of the differential susceptibility hypothesis is that it effectively addresses the "adversity bias" of the diathesis-stress model. According to the differential susceptibility hypothesis, susceptible individuals are more adversely affected by negative environments (e.g., an abusive or neglectful family), but they also benefit more from positive environments (e.g., a nurturing family). An additional advantage of the differential susceptibility hypothesis is that it offers a resolution of sorts to the debate over whether individual differences reflect phenotypic plasticity or alternative phenotypes, as well as an explanation for individual differences in within-person variability (Fleeson, 2001). Rather than supporting one or the other viewpoint, the differential susceptibility hypothesis implies some individuals are more plastic than others – that plasticity itself is an alternative phenotype (see Plaistow, Johnstone, Colegrave, & Spencer, 2004).

Belsky and Pluess (2009) offer some compelling support for their hypothesis by reevaluating data from previous diathesis-stress research, particularly research on the serotonin transporter promoter region polymorphism (5-HTTLPR). Belsky and Pluess cite the 5-HTTLPR as a potential "plasticity gene" because one variant – the long (*l*) allele – appears to be associated with lower susceptibility/plasticity and another – the short allele (*s*) – appears to be associated with higher susceptibility/plasticity. Still, the differential susceptibility hypothesis has not been rigorously tested. Furthermore, in order to determine its viability as an evolutionary model of individual differences, tests of the differential susceptibility hypothesis should also demonstrate that susceptible individuals exhibit greater *adaptive* plasticity.

## TESTING THE DIFFERENTIAL SUSCEPTIBILITY HYPOTHESIS

The primary test of differential susceptibility is whether or not individuals with proposed susceptibility genes are differentially reactive to "positive" and "negative" environmental conditions. However, many researchers, Belsky and Pluess (2009) included, have focused on broad categories of environmental factors, such as "stressful life events." They often do not distinguish between non-social stressors (or adaptive problems), such as illness, famine or drought, harsh climate, predation, or other basic threats to survival, and social stressors (or adaptive problems), let alone distinguish between different types of social stressors, such as intrasexual competition, conflict with kin, and social exclusion. As a result, it is unclear which factors are contributing to any observed gene-environment interaction effects. In addition, researchers have tended to measure broad outcome variables that may only be indirectly related to both the environmental factors being studied and the mechanism(s) by which the gene exerts its effects. From an evolutionary perspective, it may be more informative to test the differential susceptibility hypothesis by examining specific adaptive problems and the evolved neurophysiological, behavioral, cognitive, and/or affective mechanisms designed to solve them (e.g., Buss, 1995). To this end, tests of the differential susceptibility may be informed by endocrinological research, which investigates people's hormonal responses to specific positive and negative scenarios (e.g., win versus loss) in evolutionarily-relevant social contexts (e.g., status competition).

The concept of differential susceptibility can also be taken a step further. If we are to take a truly evolutionary perceptive, rather than testing whether or not "susceptible" individuals develop a "maladaptive" condition (e.g., depression), the impetus should be on demonstrating that susceptible individuals show adaptive (functional) shifts in behavior and psychology in response to the environment, whereas

the behavior and psychology of non-susceptible individuals should be less contingent upon the environment. Therefore, a test of the differential susceptibility hypothesis should not only demonstrate differential reactivity to the environment but, also, demonstrate the use of conditional behavioral and psychological strategies in susceptible but not non-susceptible individuals. To this end, one might examine behavioral and psychological responses to social feedback that have previously been identified as adaptive, such as adaptive shifts in mating psychology (see Schmitt, 2005).

Thus, the purpose of this study was to test the differential susceptibility hypothesis in a specific, evolutionarily-relevant context. Specifically, I examined differences in men's hormonal (i.e., testosterone), behavioral (e.g., approach/avoidance behavior), and psychological responses (e.g., shifts in mate preferences) to social status as a function of the 5-HTTLPR.

# Chapter II: Literature Review and Present Study

## THE 5-HTTLPR: RISK FACTOR OR PLASTICITY GENE?

Serotonin is a monoamine neurotransmitter with many important regulatory functions in the brain. Serotonin neurons project from the ralphe nuclei in the brainstem to multiple cortical and subcortical structures, including the amygdala, which processes emotional stimuli and regulates responses to threat (Adolphs, 2002), and the hypothalamus, which regulates endocrine hormones like cortisol and testosterone (Fuller, 1996). Multiple serotonin genes have been identified, including the serotonin transporter gene (*SCL6A4*), which codes for the serotonin transporter protein (5-HTT). The 5-HTT removes serotonin from the synaptic cleft by reuptaking it into the presynaptic neuron, stopping serotonin transmission and recycling it for future transmission.

Importantly, the *SCL6A4* contains a promoter region polymorphism, the 5-HTTLPR, with two common alleles: a long (*l*) allele and a short (*s*) allele (though, researchers have discovered two variants of the *l* allele, $l_A$ and $l_G$, with the $l_G$ variant functioning more like the *s* allele, e.g., Beevers, Wells, Ellis, & McGeary, 2009; Hu et al., 2005). The frequencies of these alleles vary by race and ethnicity. For example, the frequency of the *s* allele among Caucasian Americans is about 40% (e.g., Hallikainen et al., 1999; Lesch et al., 1996), among African American about 25% (e.g., Gelernter, Kranzler, & Cubells, 1997; Nellissery et al., 2003), among Asians about 80% (e.g., Kumakiri et al., 1999; Kweon, Lee, Lee, Lee, & Pae, 2005; Gelernter et al., 1997; Murakami et al., 1999; Way & Taylor, 2010), and among Croats and Russians about 15-20% (Noskova et al., 2008). The 5-HTTLPR affects 5-HTT mRNA transcription rates: $l_A$ homozygotes show higher mRNA transcription, whereas those with at least one copy of the *s* or $l_G$ allele (sometimes collectively referred to as *s'*) show lower transcription (Heils et al., 1996; Lesch et al., 1996). A lower transcription rate means fewer serotonin

transporter proteins. In effect, this reduces reuptake and increases extracellular levels of serotonin. Reduced reuptake may also decrease serotonin turnover, which could limit subsequent serotonin availability, alter serotonin receptor sensitivity, and affect signal transmission between serotonin neurons and the brain regions they innervate (David et al., 2005; Holmes, Murphy, & Crawley, 2003).

Researchers' interest in the 5-HTTLPR, particularly the *s* allele, increased after Caspi et al. (2003) found that carriers of the *s* allele who experienced more stressful events during their lifetime were more likely to develop depression. Since then, other researchers have also found that *ss* individuals who experienced more childhood adversity, peer victimization, or adulthood adversity had a greater risk of depression than *l*-carriers (Benjet, Thompson, & Gotlib, 2010; Taylor et al., 2006; Wilhelm et al., 2006; Zalsman et al., 2006). In addition, Kendler, Kuhn, Vittum, Prescott, and Riley (2005) found this genotype-environment effect was greatest at low to mild threat levels, supporting the notion that the *s* allele is related to greater sensitivity to stress. Because these findings were in line with the diathesis-stress model, researchers have considered *scl6a4* a "vulnerability gene," or risk factor, for depression. However, other studies have not always supported this conclusion (Burmeister, McInnis, & Zollner, 2008). For instance, Eley et al. (2004) found a genotype-environment effect only for females. Also, when Brummett et al. (2008) compared men and women who were caregivers for relatives with dementia (high stress condition) to non-caregivers (low stress condition), they found that depressive symptoms were highest in female caregivers with the *ss* genotype and in male caregivers with the *ll* genotype. Still other studies have failed to find any genotype-environment interaction (e.g., Fisher et al., 2012; Grassi et al., 2010; Power et al., 2010; Surtees et al., 2006; Vinberg, Mellerup, Andersen, Bennike, & Kessing, 2010).

Researchers have also attempted to link the *s* allele to other disorders, though with similarly mixed results. For instance, they have found that the *ss* genotype is more common among those with obsessive-compulsive disorder (Perez, Brown, Vrshek-Schallhorn, Johnson, & Joiner, 2006) and borderline personality disorder (Maurex, Zaboli, Ohman, Asberg, & Leopardi, 2010). In addition, two meta-analyses suggest that *s*-carriers are slightly more at risk of developing alcohol dependence (Feinn, Nellissery, & Kranzler, 2005; McHugh, Hofmann, Asnaani, Sawyer, & Otto, 2010). However, other studies have either found no genotype effect (e.g., Lee, Choi, Han, Kim, & Joe, 2009) or found that the $l_A$ allele, not the $l_G$ or *s* alleles, is associated with greater alcohol use and abuse (e.g., Gokturk et al., 2008; Hinckers et al., 2006; Hu et al., 2005; Kweon et al., 2005). One study even found a genotype-environment effect for $l_A$ homozygotes: $l_A$ $l_A$ individuals who experienced more childhood adversity and/or recent stress reported more frequent binge-drinking (Laucht et al., 2009).

Despite these mixed findings, the diathesis-stress model has continued to provide the conceptual foundation for most research on the 5-HTTLPR and has prompted much research into the mechanism(s) by which the *s* allele makes one vulnerable to developing mental disorders under stress or adversity. Some researchers have examined the relationship between the *s* allele and personality traits that have also been linked to a higher risk of depression or anxiety disorders, like neuroticism, which describes an individual's emotional stability and tendency to experience depressed mood and anxiety. Like *ss* individuals, individuals who score high in neuroticism (or negative emotionality) are more likely to develop depression and anxiety disorders, especially when they have experienced stressful life events (e.g., Kercher, Rapee, & Schniering, 2009; Lakdawalla & Hankin, 2008; Yao, Luo, Yang, Wang, & Zhu, 2009). Therefore, it has been hypothesized that carrying the *s* allele contributes to higher neuroticism, which then

makes *s*-carriers more vulnerable to developing mental disorders. However, whether or not there is a relationship between genotype and neuroticism seems to depend on the type of personality inventory used (Kumakiri et al., 1999; Schinka, Busch, & Keene-Robichaux, 2004; Schmitz, Hennig, Kuepper, & Reuter, 2007; Umekage et al., 2003), gender (Vormfelde et al., 2006), and age (Harro et al., 2009), suggesting that personality traits like neuroticism may be too complex to detect genotype effects.

Besides personality, researchers have examined more specific "abnormalities" in *s*-carriers' emotional, cognitive, endocrinological, and neurological responses to negative emotion and stress. In general, *s*-carriers are more emotionally reactive (Maurex et al., 2010) and risk averse (Whisman, Richardson, & Smolen, 2011). They have difficulty disengaging their attention from emotional stimuli (Beevers et al., 2009, p. 670) and show greater vigilance for threats (Osinsky et al., 2008), angry faces (Johnson, Gibb, & McGeary, 2010; Perez-Edgar et al., 2010), and anxiety-related words (Beevers, Gibb, McGeary, & Miller, 2007). Individuals with the *ss* genotype also show a greater increase in cortisol in response to stressful tasks (e.g., serial subtraction task, Gotlib, Joormann, Minor, & Hallmayer, 2008; public speaking task, Way & Taylor, 2010). These differences in stress response even appear to be present at birth, as infants who are homozygous for the *s* allele show a significantly higher cortisol response to a heel prick (Mueller, Brocke, Fries, Lesch, & Kirschbaum, 2010). Finally, brain imaging studies show that, compared to *ll* individuals, *s*-carriers show greater amygdala activity in response to public speaking tasks and to sad, fearful, and angry facial expressions (Dunnlowski et al., 2010; Furmark et al., 2004; Hariri et al., 2002; Hariri et al., 2005; Way & Taylor, 2010); and *s*-carriers show reduced connectivity between the amygdala and regions of the prefrontal cortex that would otherwise regulate emotional and

behavioral responses by providing inhibitory feedback to the amygdala (see Hariri & Holmes, 2006).

Nevertheless, the limitation to all of these studies is their conceptual foundation: the diathesis-stress model. As a result, researchers have focused on how people respond to negative experiences (e.g., stressful life events) and aversive stimuli (e.g., negative emotions), while overlooking the possibility (as well as data that indicate) that "vulnerable" individuals may actually respond better under positive conditions (Belsky & Pluess, 2009). For example, although Taylor et al. (2006) did find that *ss* individuals, relative to *l*-carriers, showed more depressive symptomatology if they had experienced childhood or recent adversity, their data also reveal that *ss* individuals showed significantly *fewer* depressive symptoms if they had not. Similarly, Eley et al. (2004) found that females with the *ss* genotype in a "high risk" family environment were more likely than *ll* individuals to develop depression, but in a "low risk" family environment, they were less likely than *ll* individuals to develop depression. Then, more recently, Verschoort and Markus (2011) found that college students with the *ss* genotype, compared to those with the *ll* genotype, experienced more negative affect the day of an exam (i.e., high stress day) but less negative affect on a non-exam day (i.e., low stress day). These results cannot be explained by the diathesis-stress model but, rather, lend support to the differential susceptibility hypothesis.

Support for the differential susceptibility hypothesis can also be found in one of the few direct tests conducted by Pluess, Belsky, Way, and Taylor (2010). They found no association between life events and neuroticism among *ll* individuals, but *ss* individuals who reported more negative current life events scored higher on neuroticism, and those who experienced more positive events scored lower. Still, "current life events" encompasses a broad range of social and non-social environmental factors,

making it unclear which factors are contributing to the observed effect. In addition, as previously discussed, the relationship between the *s* allele and neuroticism is unclear, perhaps because personality traits are complex and polygenic (e.g., Comings et al., 2000), making it difficult to discern the effects of a single gene. Therefore, in order to further test the prediction that "susceptible" individuals are differentially responsive to "positive" and "negative" environmental factors, it may be beneficial to examine specific evolutionarily-relevant contexts, as well as outcome variables that are more directly influenced by serotonergic function, such as endocrine hormones, which are known to influence how people approach and respond to specific (and evolutionarily relevant) social contexts, such as social status (Salvador, 2011).

### DIFFERENTIAL REACTIVITY AND TESTOSTERONE RESPONSE

Testosterone is an androgen hormone produced by the gonads (i.e., the testes in males and the ovaries in females), which are regulated by the hypothalamus-pituitary-gonadal (HPG) axis. The release of gonadotropin-releasing hormone (GnRH) from the hypothalamus causes the anterior pituitary gland to secrete luteinizing hormone (LH) into the bloodstream, which in turn, stimulates androgen production in the gonads.

Early research on testosterone focused primarily on its role in aggression. In animals, it is clear that testosterone causes aggressive behavior (see Archer, 1991). In humans, however, the causal link is less certain. Early studies found positive correlations between testosterone levels and aggression. For instance, violent crimes (e.g., homicide, rape) but not non-violent crimes (e.g., burglary, drug use) were associated with higher testosterone in both male and female inmates, and higher testosterone inmates were more likely to assault other inmates (Brooks & Reddon, 1996; Dabbs, Carr, Frady, & Raid, 1995; Dabbs, Frady, Carr, & Besch, 1987; Dabbs & Hargrove, 1997; Dabbs, Ruback,

Frady, Hopper, & Sgoutas, 1988). Nevertheless, meta-analyses show only a weak positive relationship between testosterone and aggression (Archer, 1991; Book, Starzyk, & Quinsey, 2001).

Recent research suggests that testosterone is actually more closely related to dominance than aggression, where dominance implies the intent to gain status and aggression implies the intent to cause harm (Mazur & Booth, 1998, p. 353). Baseline testosterone is positively correlated with trait dominance (Carre, Putnam, & McCormick, 2009; Grant & France, 2001; Sellers, Mehl, & Josephs, 2007), and high testosterone individuals engage in more dominance displays and status-striving behavior (e.g., Cashdan, 1995; Grant & France, 2001; Mazur & Booth, 1998). High testosterone individuals are also more attentive to yet less fearful of angry faces, which likely signal a threat to their status, while low testosterone individuals avoid looking at angry faces, a submissive behavior (van Honk et al., 1999, 2001). Baseline testosterone can even be used to predict how people will respond to being placed in positions of high and low status. In general, people experience less physiological and emotional arousal and perform better under status conditions that "match" their testosterone levels (Josephs, Sellers, Newman, & Mehta, 2006). By contrast, individuals with high baseline testosterone who are placed in low status positions and individuals with low baseline testosterone who are placed in high status positions (both instances of status "mismatches") show marked cognitive deficits (Josephs, Newman, Brown, & Beer, 2003; Newman, Guinn Sellers, & Josephs, 2005), increased cardiac arousal, increased attention to status (Josephs et al., 2006), and lower perceived efficacy on a group task (Zyphur Narayanan, Koh, & Koh, 2009).

In addition to dominance and status-striving, testosterone influences mating behavior, particularly in men. That is, just as higher testosterone is associated with

greater status-striving, it is also associated with greater mating effort. For instance, men with high testosterone report having more sexual partners (Bogaert & Fisher, 1995; Peters, Simmons, & Rhodes, 2008), and married men with high testosterone are more likely to have extramarital affairs (Booth & Dabbs, 1993). What is more, in polygynous societies, where high status men have multiple wives (see Schmitt, 2005), polygynously married men have higher testosterone than either monogamously married men or unmarried men (Alvergne, Faurie, & Raymond, 2009; Gray, 2003). By contrast, lower testosterone is not only related to lower dominance but, also, greater parenting effort, as evidenced by lower testosterone levels in men who are in committed relationships, especially those who have children (Berg & Wynne-Edwards, 2001; Burnham et al., 2003; Fleming, Corter, Stallings, & Steiner, 2002; Gray, Kahlenberg, Barrett, Lipson, & Ellison, 2002).

Given testosterone's relationship with both dominance and mating, it makes sense that testosterone is also associated with greater reward sensitivity and risk-taking (see Montoya, Terburg, Bos, & van Honk, 2012). For example, high testosterone men risk more money in an investment game (Apicella et al., 2008) and reject low offers in an ultimatum game, even though rejecting such offers results in a receiving no money at all (Burnham, 2007). Similarly, women administered testosterone show an increased sensitivity to reward and decreased sensitivity to punishment (van Honk et al., 2004), as well as reduced fear responses (Hermans, Putman, Baas, Koppeschaar, & van Honk, 2006; van Honk, Peper, & Schutter, 2005). Ultimately, these effects may stem from testosterone's effects on the amygdala (Simerly, Chang, Muramatsu, & Swanson, 1990), as well as its effects on the signaling between the amygdala and cortical regions like the prefrontal cortex and the orbitofrontal cortex (Hermans, Ramsey, & van Honk, 2008; van

Wingen, Mattern, Verkes, Buitelaar, & Fernandez, 2010), which regulate risk-taking and sensitivity to reward and punishment.

Testosterone levels also change in response to situations that involve status, mating, and/or risk-taking (e.g., Mehta, Wuehrmann, & Josephs, 2009; Wagner, Flinn, & England, 2002). In athletic competitions, both men and women show a rise in testosterone prior to and during a game (Bateup, Booth, Shirtcliff, & Granger, 2002; Edwards & Kurlander, 2010; Edwards, Wetzel, & Wyner, 2006; Oliveria, Gouveia, & Oliveria 2009), a response that would have been highly adaptive for an ancestral human insofar as it increased his/her willingness to take (physical) risks when there was an opportunity to gain status. Similarly, testosterone rises in men after brief social interactions with attractive women (Roney, Lukaszewski, & Simmons, 2007; Roney, Mahler, & Maestripieri, 2003) and when a man's status is threatened in the presence of a woman (Saad & Vongas, 2009). Notably, however, these sorts of anticipatory rises in testosterone are observed primarily in people who are highly motivated, self-confident, or anticipate winning (Carre, Muir, Belanger, & Putnam, 2006; Mazur, Booth, & Dabbs, 1992; Salvador, Suay, Gonzalez-Bono, & Serrano, 2003). Furthermore, in anticipation of a status loss or stress, testosterone levels either do not change or actually decrease (Chatterton, Vogelsong, Lu, & Hudgens, 1997; Mazur et al., 1992; Schulz et al., 1996), a response that would have been adaptive insofar as it suppressed the motivation to take potentially costly risks or to pursue mates at inopportune times.

Testosterone levels also rise or fall as a function of competitive or other status-related outcomes. For instance, testosterone levels rise after winning a competition or achieving dominance but drop after losing a competition or being subordinated (e.g., Booth, Shelley, Mazur, Tharp, & Kittok, 1989; McCaul, Gladue, & Joppa, 1992; Rose, Bernstein, & Gordon, 1975; Oliveria et al., 2009). However, it is important to note that

the direction of testosterone change appears to depend on whether the outcome "matches" an individual's baseline testosterone (or dominance). Thus, attaining high status leads to testosterone increases and low status to testosterone decreases in individuals with high testosterone (high dominance), while the opposite is true of low testosterone individuals (Jones & Josephs, 2006; Schultheiss et al., 2005).

Of course, testosterone does not exert its effects in isolation; cortisol appears to moderate testosterone's effects (Mehta & Josephs, 2010; van Honk, Harmon-Jones, Morgan, & Schutter, 2010; Viau, 2002). Cortisol is a corticosteroid hormone produced by the adrenal cortex (part of the adrenal gland). Cortisol production is triggered by activation of the hypothalamus-pituitary-adrenal (HPA) axis by the amygdala. When stimulated, the hypothalamus releases corticotropin-releasing hormone (CRH), which causes the anterior pituitary gland to release adrenocorticotropic hormone (ACTH) into the bloodstream. ACTH then triggers the adrenal cortex to release cortisol, the primary effect of which is to increase glucose metabolism and facilitate the "fight or flight" response.

In contrast to testosterone, high baseline cortisol is associated with avoidant, submissive, and risk-averse behavior. For example, individuals with high cortisol avoid angry faces (Putnam, Hermans, & van Honk, 2004; van Honk et al., 1998), exhibit low levels of aggression (Bohnke, Bertsch, Kruk, & Naumann, 2010), and tend to be more shy or socially withdrawn (Kagan, Reznick, & Snidman, 1987; Schmidt et al., 1997). They are also less impulsive and make fewer risky decisions in gambling tasks (Takahashi, 2004; van Honk, Schutter, Hermans, & Putman, 2003). High cortisol is even associated with lower social status in men (Decker, 2000; Kapuku, Treibner, & Davis, 2002).

In addition, baseline cortisol can inhibit testosterone production by acting at all levels of the HPG axis (Aakvaag et al., 1978). As a result, testosterone levels tend to be lower under chronic stress (high cortisol) conditions (Carstensen, Amer, Amer, & Wide, 1973; Kruez, Rose, & Jennings, 1972; Monden et al., 1972). Basal cortisol can also suppress the effects of basal testosterone (see van Honk et al., 2010; Viau, 2002). For instance, although high testosterone is positively correlated with aggressive behavior (Dabbs, Jurkovic, & Frady, 1991; Popma et al., 2007; Yu & Shi, 2009), this relationship is non-significant, or even slightly negative (Dabbs et al., 1991), when cortisol levels are also high. Similarly, lower testosterone-to-cortisol ratios are linked to reduced amygdala reactivity to angry faces (Hermans et al., 2008), and high cortisol reduces status-striving behavior in high testosterone individuals (Mehta & Josephs, 2010). Finally, cortisol moderates testosterone responses to both status and mating cues. For example, high baseline cortisol predicts a decrease testosterone after losing a competition (Mehta & Josephs, 2006), particularly when basal testosterone is also high (Mehta & Josephs, 2010). Then, although men's testosterone levels tend to rise after interacting with potential mates, high cortisol tends to blunt this response (Roney, Simmons, & Lukaszewski, 2010).

In sum, past endocrine research suggests that testosterone is differentially responsive to being placed in status positions (either through competitive victory/defeat or by conferral) that either match or mismatch baseline testosterone or dominance, and that testosterone responses can be further moderated by baseline levels of behavioral approach/avoidance, as measured by basal cortisol. However, given that the serotonergic system, HPA axis, and HPG axis share similar brain structures (e.g., the amygdala), it stands to reason that genetic differences in serotonergic function should influence endocrine function. As a result, the 5-HTTLPR may interact with baseline hormone

levels to influence endocrine reactivity, and the patterns of testosterone response observed in past endocrine research may be more true of *ss* individuals, while *l*-carriers are less reactive. In support of a genotype-hormone interaction, Josephs et al. (in press) recently found that baseline testosterone in *s*-carriers, not *ll* individuals, was positively associated with cortisol response to social status threats. Although Josephs et al. tested cortisol, not testosterone, response and only examined status threats, their results suggest that using similar experimental manipulations (e.g., manipulating social status) and measuring hormone responses may provide a more direct test of differential susceptibility. Thus, according to the differential susceptibility hypothesis, *ss* individuals should show greater testosterone reactivity to social status cues, either positive or negative, while *l*-carriers should show relatively blunted hormonal responses. Moreover, their responses should be moderated by baseline cortisol.

### PLASTIC *AND* NON-PLASTIC PHENOTYPES

While differential testostereone response would demonstrate differential susceptibility, it would also be useful to demonstrate that "susceptible" individuals exhibit behavioral and psychological plasticity, while non-susceptible individuals do not. In previous 5-HTTLPR research, and even in recent differential susceptibility studies, the goal has usually been to demonstrate the presence or absence of a negative outcome variable (e.g., depression or negative affect) in response to different environmental conditions. However, if susceptibility is synonymous with plasticity, then susceptible (plastic) genotypes should produce different *adaptive* phenotypes in response to different environmental conditions. Moreover, because susceptibility requires greater (and more costly) sensory sensitivity and physiological reactivity (DeWitt et al., 1998), the

phenotypes produced (or strategies employed) should tend to minimize or compensate for the costs of plasticity.

One way to test these assumptions is to examine behavioral responses to social feedback. For example, for *ss* individuals, being socially accepted by one's peers should trigger affiliative/cooperative behavior, which helps form and maintain the types of social bonds that prevent future conflict or exclusion (Baumeister & Leary, 1995). On the other hand, being criticized, derogated, or bullied by one's peers should trigger social withdrawal, which is adaptive for a susceptible individual insofar as it removes the individual from the source of conflict and prevents or minimizes the costs of chronic social stress (e.g., immunosuppression, see Segerstrom & Miller, 2004). By contrast, affiliative/withdrawal behavior in *l*-carriers should not vary as a function of social interactions; that is, an *l*-carrier may be more consistently extroverted or consistently reserved across social situations. In the context of social status, *ss* individuals should be more motivated to assume a high status position when it is conferred upon them, but when their status is taken away, they should be more motivated to behave submissively toward or withdraw from those who defeated them. By contrast, *l*-carriers should behave more consistently dominant or consistently submissive regardless of the status conferred to them by their peers.

There may also be psychological changes as a function of social feedback that would have had significant implications for fitness in the ancestral environment. In particular, because men's status was (and often still is) closely related to their mating success (see Hopcroft, 2006), one might expect changes in status to affect the mating psychology of men with susceptible genotypes. This would be an interesting addendum to current mating research, as evolutionary psychologists have often debated about whether individual differences in mating psychology reflect conditional or alternative

strategies (Gangestad & Simpson, 2000; Gross, 1996). That is, some argue that men's mating psychology should depend on factors like their social status and self-perceived mate value (see Schmitt, 2005), while others argue that men are genetically predisposed to either a short-term mating psychology – the desire for multiple mating opportunities with multiple women without investing time, energy, or resources into raising offspring – or a long-term mating psychology – the desire to form a long-term bond with one mate and invest time, energy, and resources in offspring (Bailey, Kirk, Zhu, Dunne, & Martin, 2000; Rowe et al., 2007). However, the differential susceptibility hypothesis suggests that men with susceptible genotypes should demonstrate conditional shifts in mating psychology relative to their social status, whereas *l*-carriers should not. For instance, *ss* men, not *l*-carriers, should desire more mates and be more confident approaching potential mates following a status gain, similar to the men in Surbey and Brice's (2007) study, who increased in desire for casual sex partners after experiencing an increase in self-perceived mate value.

Similarly, social status changes should influence the qualities *ss* men desire in mates. In general, men value physical attractiveness because it was a reliable and discernible cue to a woman's age, health, and fertility (e.g., Buss, 1987, 1989; Cunningham, 1986; Cunningham, Roberts, Barbee, Druen, & Wu, 1995; Singh, 1993; Symons, 1979); however, men especially prioritize attractiveness in short-term mates because, in a low investment/commitment relationship, a woman's fertility was very important to a man's mating success (Li, Bailey, Kenrick, & Linsenmeirer, 2002; Symons, 1979). For long-term mates, attractiveness is still important, but other traits, such as intelligence, kindness, emotional stability, and sense of humor, become more important because such traits influence relationship stability and the quality of the offspring the man is going to be investing in (Kenrick, Groth, Trost, & Sadalla, 1993).

Thus, when asked to consider the minimum acceptable criteria for a casual sexual relationship (e.g., one-night stand), men significantly lower their standards for traits like intelligence, kindness and understanding, and emotional stability, while maintaining high standards for attractiveness (Kenrick, Sadalla, Groth, & Trost, 1990). Conversely, when asked to consider the minimum acceptable criteria for a date or marriage partner, men have high standards not only for physical attractiveness but, also, for intelligence, kindness and understanding, and emotional stability (Kenrick et al., 1990).

Nevertheless, because high status men do not have trouble attracting mates (e.g., Buss, 1994; Grammar, 1992; Hill & Hurtado, 1996), they can afford to be more selective, even when selecting short-term mates. Low status men, on the other hand, may have more difficulty attracting mates; therefore, they need to lower their standards, particularly for casual sex partners, in order to increase their mating opportunities. If *ss* men are more sensitive to changes in their status, then they should be more selective after a status gain and less selective after a status loss. That is, *ss* individuals elevated to a high status position should raise their standards for a one-night stand, while *ss* carriers demoted to a low status position should lower their standards. The standards of *l*-carriers, on the other hand, should not be affected by changes in status.

## PRESENT STUDY

The purpose of the present study was to test the differential susceptibility hypothesis in a specific, evolutionarily-relevant context by examining individual differences in men's hormonal, behavioral, and psychological responses to social status cues as a function of the 5-HTTLPR.

In this study, groups of 3-4 male participants socialized for 5-10 minutes before they were told that the study was part of a larger initiative to create a student-run Honor

Committee. Participants used "ballots" to nominate one person in the group who they believed would make the best committee leader and one person they believed was the weakest (i.e., not good for an honor committee). Then, participants were separated into private rooms, where they were told either everyone voted them leader or everyone voted them weakest. Finally, participants responded to a series of mating questionnaires before being given the choice to examine an actual honor violation case either with the committee or alone.

To demonstrate differential susceptibility, two criteria must be met. First, the susceptibility factor must be independent of both the environmental factor and the outcome (Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007). In this study, social status threat or confirmation (i.e., the environmental factor) was experimentally controlled and, therefore, independent of genotype. Still, the 5-HTTLPR should not be independently associated with hormonal, behavioral, or psychological responses:

> *Prediction 1*: There will be no main effects of genotype predicting any of the outcome variables (i.e., hormone response, decision to participate on the committee or work alone, and responses to mating questionnaires).

The second criterion for demonstrating differential susceptibility is a cross-over interaction; that is, the "slope for the susceptible subgroup should be significantly different from zero and at the same time significantly steeper than the slope for the non- (or less) susceptible subgroup" (Belsky & Pluess, 2009, p. 888). In terms of testosterone response, this means that *ss* individuals, not *l*-carriers, should not only be more hormonally reactive but, also, show differential testosterone responses to vote feedback. Furthermore, the testosterone responses of *ss* individuals should be moderated by basal

cortisol because basal cortisol is associated with approach/avoidance behavior (Kagan et al., 2003; Shoal, Giancola, & Kirillova, 2003).

Specifically, after being voted leader, *ss* individuals with high basal cortisol should decrease in testosterone because high basal cortisol is associated with avoidant behavior. On the other hand, *ss* individuals with low basal cortisol should increase in testosterone after being voted leader because low basal cortisol is associated with approach behavior.

By contrast, after being voted weakest, I expect to see the opposite pattern: *ss* participants with high basal cortisol should increase in testosterone because high basal cortisol is associated with avoidant behavior. On the other hand, *ss* individuals with low basal cortisol should decrease in testosterone after being voted weakest because low basal cortisol is associated with approach behavior.

*Prediction 2a*: *l*-carriers should show blunted testosterone responses that are independent of vote feedback.

*Prediction 2b*: *ss* individuals should show greater changes in testosterone, the direction of which should be dependent upon vote feedback.

*Prediction 2c*: Vote feedback should determine testosterone change among *ss* individuals depending on basal cortisol level.

Finally, in addition to satisfying the criteria for demonstrating differential susceptibility, this study was designed to test whether the *s* allele is associated with behavioral and psychological plasticity:

*Prediction 3a*: *l*-carriers' decisions to participate in the committee or work alone, as well as their responses the mating questionnaires, will not be affected by vote feedback.

*Prediction 3b*: *ss* individuals should be more likely to choose to participate on the committee after being voted leader but more likely to choose to work alone after being voted weakest.

*Prediction 3c*: After being voted leader, *ss* individuals should be less anxious about approaching a potential mate and increase their standards for traits like intelligence and emotional stability when considering a potential one-night stand. By contrast, after being voted weakest, *ss* individuals should be more anxious about approaching a potential mate and lower their standards for traits like intelligence and emotional stability when considering a potential one-night stand.

# Chapter III: Methodology

## PARTICIPANTS

Participants were 119 heterosexual male undergraduates at the University of Texas at Austin. Participants were students in introductory psychology courses and received course credit for their participation. Because the frequency of the *s* allele varies by race/ethnicity (e.g., Hallikainen et al., 1999; Kweon et al., 2005; Nellissery et al., 2003), only Caucasians of either non-Hispanic or Hispanic decent were recruited for this study; however, when asked to confirm their ethnicity, seven participants did not identify themselves as Caucasians of either non-Hispanic or Hispanic decent. Thus, the data from these participants were excluded from all analyses, leaving a total of 111 participants (88 non-Hispanic and 23 Hispanic) with a mean age of 20.05 years (*SD* = 1.52). In addition, 20 participants had at least one salivary hormone measurement with a coefficient of variation (CV) greater than 15 %; therefore, the data from these participants were excluded from any analyses that included hormone measurements. The total sample for analyses using hormone measurements was 91 participants (70 non-Hispanic and 21 Hispanic) with a mean age of 20.00 years (*SD* = 1.57).

## BACKGROUND QUESTIONNAIRES

### Demographics Questionnaire

Participants reported their birth date, sex, height, ethnicity, socioeconomic status of origin, sexual orientation, and relationship status (see Appendix A).

### Center for Epidemiologic Studies Depression Scale (CES-D)

The CES-D (Radloff, 1977) is a 20-item questionnaire that measures depressive symptoms. Participants reported how often they have felt various symptoms, such as feeling restless or lonely, over the past week using a 4-point scale, where 0 = “rarely or

none of the time (<1 day)" and 3 = "most or all of the time (5-7 days)." Higher aggregate scores indicate more depressive symptoms; scores over 15 are generally considered indicative of depression. In this study, the Cronbach's alpha for this scale was .913.

**Rosenberg Self-Esteem Scale (RSES)**

The RSE scale (Rosenberg, 1965) was designed as a measure of global self-esteem. It consists of 10 items related to overall feelings of self-worth or self-acceptance. Using a 4-point scale, participants reported whether they strongly agree, agree, disagree, or strongly disagree with statements such as "On the whole, I am satisfied with myself" and "I feel that I have a number of good qualities." Higher aggregate scores indicate higher self-esteem. In this study, the Cronbach's alpha for this scale was .681.

**State-Trait Anxiety Inventory (STAI)**

The STAI (Spielberger, Gorusch, & Lushene, 1970) is a two-part questionnaire, each part consisting of 20 statements, that measures situational, state-related anxiety levels and stable, trait-related anxiety levels. For this study, we only used the trait anxiety scale, which asks participants to report how often they *generally* experience certain emotional states, such as feeling nervous or having disturbing thoughts, using a 4-point scale (where 1 = "almost never" and 4 = "almost always"). Higher aggregate scores indicate higher trait anxiety. In this study, the Cronbach's alpha for this scale was .879.

**Highly Sensitive Person Scale (HSPS)**

The HSPS (Aron & Aron, 1997) consists of 27 statements and is used to measure sensory-processing sensitivity, or a person's sensitivity to subtle stimuli and tendency to be overwhelmed by stimulation. Participants rated how true each statement is of them using a 7-point scale (where 1 = "not at all true" and 7 = "extremely true"). Examples of

statements include “I tend to be more sensitive to pain” and “I startle easily.” Higher average scores indicate greater sensitivity and are associated with higher perceived stress and poorer health (Benham, 2006). Nevertheless, while the HSPS was originally believed to measure a single construct (Aron & Aron, 1997), subsequent research has identified three subscales: Ease of Excitability (EOS,12 items), which includes items such as “I get rattled when I have a lot to do in a short amount of time;” Aesthetic Sensitivity (AES, 6 items), which includes items such as “I have a rich, complex inner life;” and Low Sensory Threshold (LST, 7 items), which includes items such as “I am particularly sensitive to the effects of caffeine” (Smolewska, McCabe, & Woody, 2006). In the present study, the Cronbach’s alphas for these three subscales were .821, .676, and .838, respectively. The Cronbach’s alpha for the entire scale was .885.

**Big Five Inventory (BFI)**

The BFI (John & Srivastava, 1999) consists of 44 self-descriptive statements (e.g., “I see myself as someone worries a lot” or “I see myself as someone who is talkative”). Participants rated how much they agree with each statement using a 5-point scale (where 1 = “disagree strongly” and 5 = “agree strongly”). The BFI measures the Big Five factors of personality: Neuroticism (8 items), Extroversion (8 items), Openness to Experience (9 items), Agreeableness (9 items), and Conscientiousness (9 items). Higher average scores indicate higher levels of a given factor. In this study, the Cronbach’s alphas for each factor were .807, .825, .813, .803, and .729, respectively.

**Sociosexual Orientation Inventory Revised (SOI-R)**

The SOI-R (Penke & Asendorpf, 2008) is a 9-item questionnaire used to assess the degree to which a person prefers casual, short-term sexual relationships (i.e., unrestricted orientation) or committed, long-term sexual relationships (i.e., restricted

orientation). The SOI-R consists of three subscales (three items each): Behavior, which refers to how often one engages in casual sex; Attitude, which refers to one's attitudes toward casual sex; and Desire, which refers to how often a person experiences sexual arousal around strangers (i.e., potential casual sex partners). Global SOI is calculated by averaging the three subscales. Higher average scores indicate a more unrestricted orientation. The Cronbach's alphas for the three subscales were .827, .873, and .835, respectively. The Cronbach's alpha for Global SOI was .692.

**Personality Research Form (PRF)**

The PRF (Jackson, 1984) consists of over 300 true-false statements and measures multiple aspects of personality (e.g., achievement, autonomy, harm avoidance). For the purpose of this study, we only used the Dominance subscale, which asks participants to respond "true" or "false" to 16 statements, such as "The ability to be a leader is very important to me" and "I would make a poor military leader" (reverse scored). Higher aggregate scores indicate a more dominant personality. In this study, the Cronbach's alpha for this scale was .937.

**Social Dominance Orientation Scale (SDO)**

The SDO scale (Pratto, Sidanius, Stallworth, & Malle, 1994) is designed to measure preference for inequality between groups. Participants use a 7-point scale (where 1 = "disagree strongly" and 7 = "agree strongly") to rate how much they agree with 16 statements, such as "Some groups of people are simply inferior to other groups" and "No group should dominate in society" (reverse scored). Higher average scores indicate a preference for hierarchy and inequality. In this study, the Cronbach's alpha for this scale was .909.

**PAD Trait Dominance Scale (PAD-TDS)**

The PAD-TDS (Mehrabian & Hines, 1978) is a 26-item measure of an individual's awareness of his or her dominance and submissive behavior. Using a 9-point scale (where -4 = "very strong disagreement" and +4 = "very strong agreement"), participants rated how much they agree with statements like "I control others more than they control me" and "I usually question rules and regulations." Higher aggregate scores indicate higher dominance and a greater sense of control. Trait dominance is negatively correlated with sensitivity to rejection (Mehrabian, 1994) and conformity (Mehrabian & Stefl, 1995). In this study, the Cronbach's alpha for this scale was .917.

**Buss-Perry Aggression Scale**

The Buss-Perry Aggression Scale (Buss & Perry, 1992) is a 29-item measure that asks participants to respond to statements, such as "If somebody hits me, I hit back" and "I have trouble controlling my temper," using a 7-point scale (where 1 = "Extremely uncharacteristic of me" and 7 = "Extremely characteristic of me"). This scale assesses four aspects of aggression: Physical Aggression (9 items), Verbal Aggression (5 items), Anger (7 items), and Hostility (8 items). Higher aggregate scores indicate higher aggression. For this study, the Cronbach's alphas for the subscales were .691, .636, .740, and .871, respectively. The Cronbach's alpha for the entire scale was .908.

**Leadership Behavior Description Questionnaire Form XII (LBDQ-XII)**

The LBDQ-XII (Stogdill, 1963) is a 100-item questionnaire designed to assess 12 patterns of leadership behavior: Representation (i.e., "speaks and acts as the representative of a group"), Reconciliation (i.e., "reconciles conflicting demands and reduces disorder"), Tolerance of Uncertainty (i.e., "is able to tolerate uncertainty and postponement without anxiety or upset), Persuasiveness (i.e., "uses persuasion and

argument effectively"), Initiation of Structure (i.e. "clearly defines own role, and lets followers know what is expected"), Tolerance and Freedom (i.e., "allows followers to scope for initiative, decision and action), Role Assumption (i.e., "actively exercises the leadership role"), Consideration (i.e., "regards the comfort, well being, status, and contributions of followers"), Production Emphasis (i.e., "applies pressure for productive output"), Predictive Accuracy (i.e., "exhibits foresight"), Integration (i.e., "maintains closely knit organizations; resolves intermember conflicts"), and Superior Orientation (i.e., "maintains cordial relations with superiors; has influence with them; is striving for higher status") (Stodgill, 1963, p.3). In the present study, the Cronbach's alphas for these subscales ranged from .508 to .762, with the exception of Reconciliation (5 items; $\alpha$ = .445) and Tolerance of Uncertainty (10 items; $\alpha$ = .332). The Cronbach's alpha for the entire scale was .923.

**Singelis Individualism-Collectivism Scale (IND-COL)**

The IND-COL (Singelis, 1994) is a 24-time scale designed to measure independent and interdependent self-construals. Participants use a 7-point scale (where 1 = "disagree strongly" and 7 = "agree strongly") to indicate the degree to which they agree with statements measuring their independent self-construals, such as "I enjoy being unique and different from others in many respects," and their interdependent self-construals, such as "It is important for me to maintain harmony within my group." Total scores on the independent subscale were subtracted from total scores on the interdependent subscale to give an overall individualism-collectivism index, where positive scores indicate a more interdependent self-construal and negative scores a more independent self-construal. In the present study, the Cronbach's alphas for the

independent and interdependent subscales (12 items each) were .783 and .871, respectively.

### Levenson Self-Report Psychopathy Scale (LSRP)

The LSRP (Levenson, Kiehl, & Fitzpatrick, 1995) was designed to measure Primary Psychopathy (e.g., arrogance, callousness) and Secondary Psychopathy (e.g., impulsivity, irresponsibility). Participants rated the degree to which they agree or disagree with 26 statements using a 4-point scale (where 1 = "strongly disagree" and 4 = "strongly agree"). An example of a statement from the primary psychopathy subscale is "Looking out for myself is my top priority." An example of a statement from the secondary psychopathy subscale is "I don't plan anything very far in advance." In the present study, the Cronbach's alpha for the 16-item primary psychopathy subscale was .813, and the Cronbach's alpha for the 10-item secondary psychopathy subscale was .731. The Cronbach's alpha for the entire scale was .864.

## MATING QUESTIONNAIRES

### Dating Anxiety

The purpose of the dating anxiety questionnaire was to examine how anxious (or confident) participants were about approaching and asking out potential romantic partners and how upset they would be if they were rejected by potential romantic partners after a social status manipulation. Participants were shown four photographs of young adult Caucasian women (two attractive and two unattractive). The photos were taken from a bank of pre-rated photographs that have been used as stimuli in previous experiments. The photos showed only the women's faces. All women had similar hair styles and were smiling. Participants were asked to imagine asking each woman to meet them or to go on a date with them. They used a 0-100 scale (where 0 = "not at all/extremely low" and 100

= “completely/extremely high”) to respond to a total of seven questions concerning their interest in meeting each woman, how anxious they would be to ask each woman out on a date, how interested each woman would be in dating them, and how upset they would be if each woman rejected them (Kugeares, 2002). (Refer to Appendix B for a complete example.) Participants who were in committed relationships at the time of the experiment were asked to respond as if they were not currently in a relationship. Independent t-tests later confirmed that there were no differences in responses to any of the questions as a function of relationship status.

Responses to questions concerning the attractive women were averaged, and responses concerning the unattractive women were averaged. Paired-samples t-tests revealed that the attractive women received significantly higher ratings for all questions except “How interested do you think she would be [in going out with you]?” In general, men were more interested in and willing to go out with the attractive women but, also, more anxious to ask them out and more upset if rejected. However, when assessing the women’s interest, attractiveness did not matter.

**Minimum Acceptable Mate Selection Criteria**

Participants used percentile ranking to describe 14 characteristics (e.g., intelligence, emotional stability, physical attractiveness, dominance) they would use in choosing a romantic partner (Kenrick et al., 1990, 1993). It was explained that the percentiles corresponded to how a person stacks up against all the people one might encounter on the street or a college campus during a typical week. Participants were given the following example: “Suppose you are a male and that your relevant population of potential mates is women. So, consider the characteristic of friendliness. If we could rank all the women by their friendliness, then the friendliest woman would be at the 100$^{th}$

percentile of friendliness – she is friendlier than 100% of all women. The most unfriendly woman is at the 0th percentile of friendliness – she is friendlier than 0% of all women. The woman at the 50th percentile of friendliness is friendlier than exactly 50% of all women and less friendly than 49% of the people on this dimension." Participants then reported the minimum percentile for each of the 14 characteristics they would find acceptable in a partner at each of three levels of relationship involvement: (a) a one-night stand, (b) a date, and (c) marriage (Kenrick et al., 1993, p. 954). Again, participants who were in committed relationships at the time of the experiment were asked to respond as if they were not currently in a relationship. Independent t-tests later confirmed there were no significant differences in response as a function of relationship status.

Participants also used percentile rankings to describe the same 14 characteristics as they pertained to themselves, where the percentiles corresponded to how the participants stack up against all the men they might encounter on the street or a college campus during a typical week. There were no significant differences in self-ratings as a function of relationships status.

### 5-HTTLPR GENOTYPING

Genomic DNA was isolated from saliva. Each participant rinsed his mouth with distilled water to remove any food particles. Then, he delivered 2-3 mL of saliva into a standard 15 mL test tube. After he delivered saliva into the tube, an "S-swab" (i.e., a sterile cotton swab pre-prepared using buffer, NaCL, SDS, and proteinase K solutions) was placed tip down into the tube. The tube was capped and shaken. Tubes were stored at 5º C until the samples were shipped for DNA extraction. DNA extraction was performed using previously published techniques (Hu et al., 2005).

Using biallelic genotyping, the genotype frequencies were: 36 *ll*, 48 *ls*, and 27 *ss*. (After removing those with insufficient hormone data, the frequencies were: 27 *ll*, 40 *ls*, and 24 *ss*.) Using triallelic genotyping, the genotype frequencies were: 23 $l_Al_A$, 53 $l_As'$ (i.e., $l_Al_G$ or $l_As$), and 35 *s's'* (i.e., $l_Gs$, $l_Gl_G$, or *ss*). (After removing those with insufficient hormone data, the frequencies were: 18 $l_Al_A$, 45 $l_As'$, and 28 *s's'.)* Preliminary analyses revealed significant effects when using biallelic but not triallelic genotyping. Furthermore, preliminary analyses did not reveal any significant gene-environment effects when comparing *ll* individuals and *s*-carriers (e.g., for testosterone response, $\beta$ = .011, $p$ = .66) or when comparing *ll*, *ls*, and *ss* individuals (e.g., for testosterone response, $\beta$ = -.016, $p$ = .39), indicating that *ll* individuals were not significantly different from *ls* individuals. Thus, an *l*-carrier group was created and all subsequent analyses compared *l*-carriers and *ss* homozygotes.

### SALIVARY TESTOSTERONE AND CORTISOL

Testosterone and cortisol levels vary depending on the time of day, with levels usually at their highest in the morning but decreasing and becoming more stable later in the day (e.g., Diver, Imitaz, Ahmad, Vora, & Fraser, 2003; Hucklebridge, Hussain, Evans, & Clow, 2005); therefore, all laboratory sessions were held between 2:00 pm and 6:00 pm. Additionally, participants were asked to avoid eating or drinking for one hour before their laboratory session. Each participant was asked to provide two saliva samples by freely releasing saliva into 15 mL collection tubes. The date and time of collection for each sample were recorded. All saliva samples were frozen at -20$^o$ C until subsequent immunoassay using Salimetrics enzyme immunoassay kits (State College, PA, USA). All samples were assayed in duplicate. The intra-assay and inter-assay CVs for both cortisol and testosterone were less than 15%.

## DESIGN AND PROCEDURE

The study consisted of two one-hour sessions – an online session and a laboratory session. Within 24 hours of signing up for a scheduled laboratory session, participants were contacted via email by the experimenter. In the email, participants were given a URL for the online session. Participants were allowed to complete the online session at their convenience, but they were required to complete the session prior to their scheduled laboratory session. This was done to minimize the questionnaires' influence on behavior in the laboratory and to prevent experiences in the laboratory from affecting responses on the questionnaires. Furthermore, to minimize order effects and confounds due to fatigue, the order in which the online questionnaires were presented and the order of the items within each questionnaire were randomized for each participant.

Laboratory sessions consisted of three to four unacquainted participants. Upon arrival, participants were separated into private rooms, where they reviewed and signed the informed consent form. Each participant then provided a saliva sample for genotyping. After the DNA sample was collected and stored (about 15 minutes after their arrival), the participants provided another saliva sample for baseline testosterone and cortisol measurements.

Next, the participants were given name tags and taken to a "common room," where they were asked to socialize with each other for 5-10 minutes. (There were a few laboratory sessions that consisted of only two participants. In those cases, a trained male confederate acted as a third participant for this part of the study.) Then, as a group, the participants were told that the study was part of a larger initiative to create a student-run Honor Committee. The participants were given "ballots" to nominate one person in the group who they believed would make the best leader of the committee. They were also asked to nominate the one person they thought was the weakest and, therefore, should not

be on the committee. (For a complete script, see Appendix C.) Participants were not allowed to discuss their nominations; instead, they filled in their ballots and returned them directly to the experimenter.

After the experimenter collected the nominations, participants returned to their private rooms, where they were left alone for a few minutes while they believed the votes were being counted. Then, they were consulted individually about the "results" of the vote. They were told one of the following:

> *Condition 1:* "Congratulations! The vote was unanimous; all of the other participants think you should be the leader of the Honor Committee."

> *Condition 2:* "I'm sorry, but the vote was unanimous; all of the other participants think you should not be the on the Honor Committee."

Next, while the participants believed the research staff were setting up the final part of the study, participants completed some "unrelated tasks" on the computers in their private rooms. These "unrelated tasks" were actually the minimum acceptable mate selection criteria and dating anxiety questionnaires. The order of the questionnaires was randomized for each participant.

After 20 minutes, a second saliva sample was collected and stored. Participants were then informed that for the remainder of the study the experimenters wanted to see how well the participants worked together as a committee in determining an outcome for an actual honor violation case that was previously reviewed by an honor committee at another university. Participants who were told they were voted to be the leader of the committee were given two choices: (1) they could participate in the committee; or (2)

they could choose not to participate and, instead, examine the case alone. Participants who were told they were voted weakest were told that they would technically have to spend the remaining time examining the case alone but that one person who was voted to be on the committee decided not to participate. So, they were given two choices: (1) they could examine the case alone; or (2) they could take the person's place and participate in the committee. After a participant made his choice, the experiment ended. The participant was informed of the true purpose of the study and debriefed.

# Chapter IV: Results

## BASELINE ANALYSES

### Genotype

Genotype (*l*-carriers vs. *ss* individuals) was independent of ethnicity (non-Hispanic vs. Hispanic), $\chi^2$ (1) = 1.72, $p$ = .19; socioeconomic status, $\chi^2$ (4) = 6.13, $p$ = .19; and relationship status, $\chi^2$ (2) = 1.99, $p$ = .37. In addition, *l*-carriers and *ss* carriers were not significantly different in age, $t(109)$ = .96, $p$ = .34, or self-reported height, $t(109)$ = .15, $p$ = .88.

Independent t-tests were conducted to compare the background questionnaire scores of *l*-carriers and *ss* individuals (see Table 1). The LBDQ-Reconciliation subscale scores of *l*-carriers and *ss* individuals were significantly different at the $\alpha$ = .05 level; *l*-carriers ($M$ = 17.38, $SD$ = 2.57) scored higher than *ss* individuals ($M$ = 16.19, $SD$ = 2.48), suggesting that *l*-carriers are better at reconciling conflict. However, after applying a Bonferroni correction ($\alpha$ = .05 / 39 = .001), this difference was no longer significant.

These results did not change when limiting the data set to only those participants with sufficient hormone data.

Table 1: Means (and standard deviations) for background questionnaire scores for *l*-carriers and *ss* individuals.

| Construct (Scale) | *ll/ls* | *ss* | *t*(109) | *p*-value |
|---|---|---|---|---|
| **Depression (CESD)** | 14.11 (11.30) | 16.89 (13.84) | -1.05 | .30 |
| **Self-Esteem (RSES)** | 37.23 (5.53) | 36.59 (4.68) | .54 | .59 |
| **Sensitivity (HSPS)** | | | | |
| Ease of Excitation (EOS) | 3.36 (.93) | 3.45 (.90) | -.47 | .64 |
| Aesthetic Sensitivity (AES) | 3.91 (.92) | 4.04 (1.15) | -.59 | .56 |
| Low Sensory Threshold (LST) | 2.43 (1.01) | 2.53 (1.24) | -.46 | .65 |
| **Trait Anxiety (STAI)** | 34.04 (11.04) | 35.85 (10.57) | -.75 | .45 |
| **Sociosexual Orientation (SOI)** | | | | |
| Behavior | 1.96 (1.26) | 2.51 (1.71) | -1.78 | .08 |

| Construct (Scale) | *ll/ls* | *ss* | *t*(109) | *p*-value |
|---|---|---|---|---|
| Attitude | 4.77 (2.35) | 5.09 (2.18) | -.62 | .54 |
| Desire | 4.87 (2.05) | 5.10 (1.63) | -.53 | .60 |
| Global | 3.87 (1.51) | 4.23 (1.52) | -1.09 | .28 |
| **Aggression (Buss-Perry)** | | | | |
| Physical | 22.13 (6.78) | 23.04 (5.84) | -.62 | .53 |
| Verbal | 14.51 (3.73) | 16.00 (3.03) | -1.88 | .06 |
| Anger | 16.01 (6.10) | 16.78 (6.27) | -.56 | .57 |
| Hostility | 20.90 (7.29) | 23.04 (8.16) | -1.28 | .20 |
| **Psychopathy (LSRP)** | | | | |
| Primary (Arrogance) | 32.86 (7.40) | 34.00 (9.67) | -.65 | .52 |
| Secondary (Impulsiveness) | 20.55 (5.17) | 22.48 (4.74) | -1.72 | .09 |
| **Social Dominance (SDO)** | 3.21 (1.09) | 3.04 (1.09) | .73 | .47 |
| **Dominance (PRF)** | 10.89 (4.16) | 12.11 (5.55) | -1.22 | .23 |
| **Dominance (PAD)** | 61.33 (23.13) | 72.07 (36.86) | -1.80 | .08 |
| **Individualism-Collectivism** | | | | |
| Independence | 56.26 (9.36) | 56.89 (11.91) | -.28 | .78 |
| Interdependence | 57.00 (11.08) | 55.96 (12.09) | .41 | .68 |
| Index | .74 (9.83) | -.93 (9.88) | .77 | .45 |
| **Personality (BFI)** | | | | |
| Extroversion | 3.28 (.70) | 3.40 (.64) | -.78 | .44 |
| Agreeableness | 3.71 (.52) | 3.76 (.66) | -.44 | .66 |
| Conscientiousness | 3.44 (.66) | 3.32 (.54) | .84 | .40 |
| Neuroticism | 2.63 (.72) | 2.45 (.68) | 1.15 | .25 |
| Openness | 3.56 (.71) | 3.51 (.55) | .28 | .78 |
| **Leadership (LBDQ)** | | | | |
| Representation | 16.26 (3.64) | 17.19 (3.10) | -1.19 | .24 |
| Reconciliation | **17.38 (2.57)** | **16.19 (2.48)** | **2.12** | **.04** |
| Tolerance of Uncertainty | 32.37 (3.53) | 32.33 (3.43) | .04 | .97 |
| Persuasion | 34.11 (4.66) | 34.37 (4.30) | -.26 | .80 |
| Structure | 35.02 (5.28) | 35.37 (4.92) | -.30 | .76 |
| Tolerance and Freedom | 35.51 (4.50) | 35.11 (4.75) | .40 | .69 |
| Role Assumption | 34.82 (5.81) | 32.89 (5.47) | 1.52 | .13 |
| Consideration | 34.44 (3.35) | 34.59 (3.97) | -.20 | .85 |
| Production Emphasis | 34.01 (5.54) | 34.19 (4.38) | -.15 | .88 |
| Predictive Accuracy | 18.07 (2.42) | 17.52 (2.78) | 1.00 | .32 |
| Integration | 18.55 (2.74) | 17.89 (2.95) | 1.07 | .29 |
| Superior Orientation | 37.23 (4.99) | 36.78 (5.81) | .39 | .70 |

*Table 1 Continued*

**Baseline Testosterone and Cortisol**

As in past research (e.g., Josephs et al., 2011; Mehta et al., 2008; Mehta & Josephs, 2010), baseline testosterone and cortisol levels were modestly positively correlated, $r = .21$, $p = .05$. Baseline testosterone was not correlated with any scores on the online questionnaires. Baseline cortisol was only negatively correlated with Extroversion, $r = -.22$, $p = .04$.

There were no significant differences between *l*-carriers and *ss* individuals in baseline testosterone, $t(89) = -.11$, $p = .91$, or baseline cortisol, $t(89) = 1.75$, $p = .08$. Also, to confirm that there were no differences in baseline hormones as a function of either genotype or condition, I ran separate 2 (Genotype: *l*-carriers vs. *ss* individuals) x 2 (Condition: voted leader vs. voted weakest) ANOVAs for baseline testosterone and baseline cortisol. There were no differences in baseline testosterone, either as a function of Genotype ($F(1, 87) = 2.24$, $p = .14$), Condition ($F(1, 87) = .01$, $p = .93$), or the Genotype x Condition interaction ($F(1, 87) = .76$, $p = .39$). In addition, there were no differences in baseline cortisol, either as a function of Genotype ($F(1, 87) = .15$, $p = .71$), Condition ($F(1, 87) = 2.97$, $p = .09$), or the Genotype x Condition interaction ($F(1, 87) = .04$, $p = .85$).

**Genotype, Baseline Hormones, and Background Questionnaires**

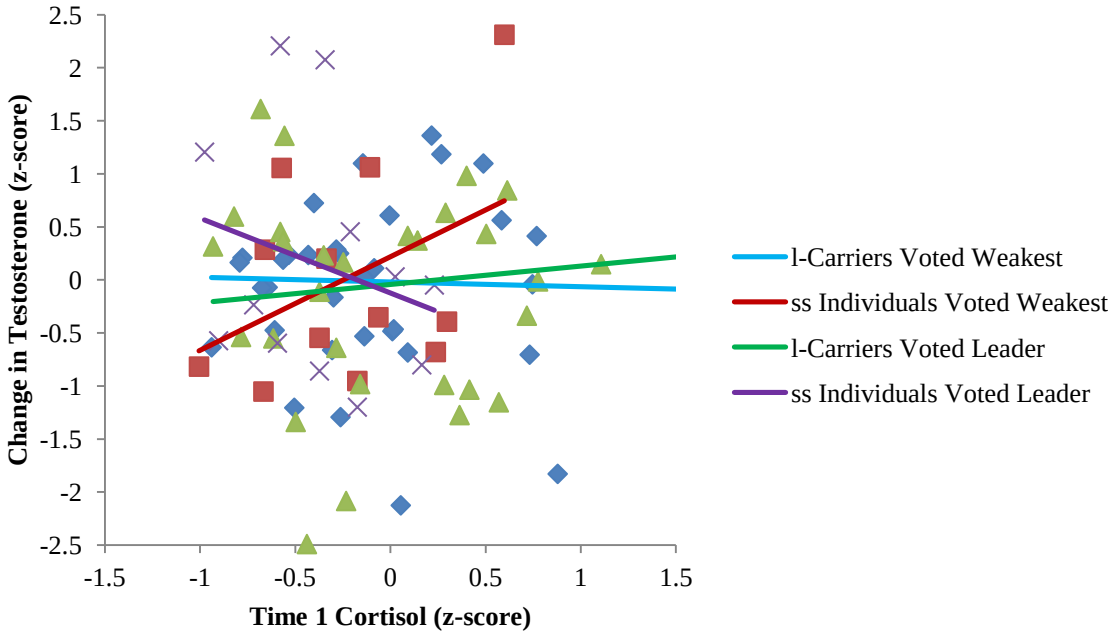
To determine whether genotype and baseline hormones together predicted traits like dominance, anxiety, and personality, baseline testosterone (Time 1 T), baseline cortisol (Time 1 C), Genotype (*l*-carriers vs. *ss* individuals), and their interactions were entered into separate linear regressions predicting scores on the online questionnaires. There were no significant Time 1 T x Time 1 C x Genotype interactions predicting online questionnaire scores. There were also no significant Time 1 T x Genotype or Time 1 C x Genotype interactions.

## TESTOSTERONE RESPONSES

It was predicted that baseline cortisol would be associated with differential testosterone responses to vote feedback in *ss* individuals but not *l*-carriers. Therefore, baseline cortisol (Time 1 C), Condition (voted leader vs. voted weakest), Genotype (*l*-carriers vs. *ss* individuals), and their interactions were entered as predictors in a linear regression model predicting time 2 testosterone. Baseline testosterone was included as a covariate.

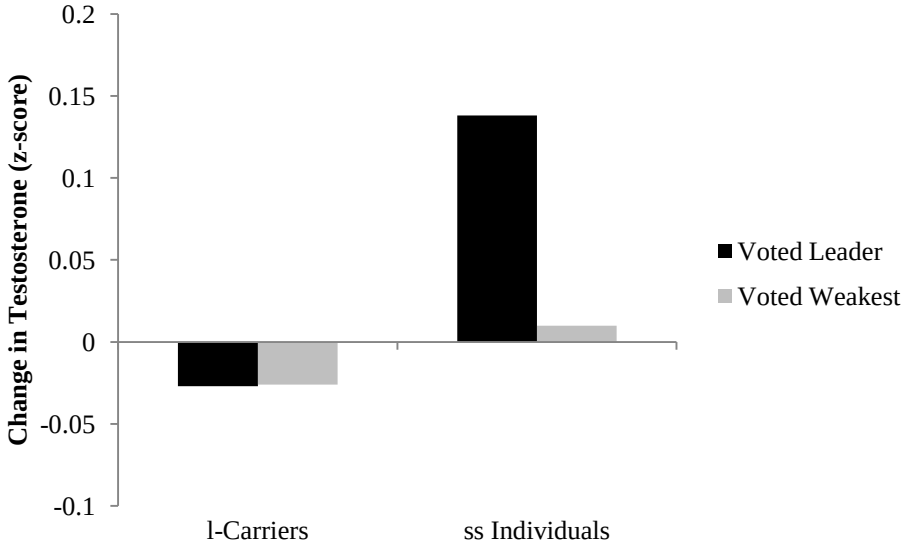
The model was significant, $R^2 = .49$, $F(8, 82) = 9.64$, $p < .001$, and there was a significant Time 1 C x Condition x Genotype interaction, $\beta = -702.52$, $p = .02$, in support of differential susceptibility. As shown in Figure 1, baseline cortisol was negatively associated with change in testosterone for *ss* individuals voted leader but positively associated with change in testosterone for *ss* individuals voted weakest. By comparison, there was relatively no association between baseline cortisol and testosterone response as a function vote feedback in *l*-carriers.

Figure 1: Change in testosterone as a function of genotype, vote feedback, and baseline cortisol.




Differential susceptibility was also supported by a significant Condition x Genotype interaction, $\beta = 107.12$, $p = .02$. As shown in Figure 2, *l*-carriers' testosterone responses did not depend on vote feedback. By contrast, *ss* individuals in the leader condition increased in testosterone, while *ss* individuals voted weakest showed almost no change in testosterone.

Figure 2: Change in testosterone as a function of genotype and vote feedback.




## BEHAVIORAL AND PSYCHOLOGICAL RESPONSES

### Decision to Participate on Committee or Work Alone

It was predicted that if *ss* individuals are differentially susceptible to positive and negative status feedback, they should have been more likely to want to participate on the honor committee (i.e., approach) after being voted leader and more likely to want to work alone (i.e., withdraw) after being voted weakest. Because participants' decisions to participate on the committee or work alone were made 20 minutes after the vote feedback manipulation, and about 35-40 minutes after the collection of baseline measurements, hormone levels were not expected to predict men's decisions to participate on the committee or work alone. Nevertheless, past research suggests that the decision to participate or work alone may be moderated by baseline testosterone and/or cortisol (e.g.,

Mehta & Josephs, 2010). Thus, testosterone (Time 1 T), baseline cortisol (Time 1 C), Condition (voted leader vs. voted weakest), Genotype (*l*-carriers vs. *ss* individuals), and their interactions were entered as predictors in a binary logistic regression model predicting decision to participate or work alone.

After removing the non-significant Time 1 C interaction effects, Time 1 T interaction effects, and Time 1 C main effect, the final model ($\chi^2$ (4) = 13.04, $p$ = .01) included a significant main effect of Time 1 T ($\beta$ = .01, $p$ = .03), a significant main effect of Condition ($\beta$ = 3.28, $p$ = .03), and a significant Condition x Genotype interaction ($\beta$ = -.3.40, $p$ = .02). The predicted probabilities of choosing to participate on the honor committee can be found in Figure 3. In general, higher testosterone was associated with a decreased probability of choosing to participate on the honor committee. More importantly, however, in support of differential susceptibility, vote feedback did not affect whether or not *l*-carriers chose to participate on the honor committee – 76% of those voted leader and 79% of those voted weakest chose to participate on the committee, $\chi^2$ (1) = .12, $p$ = .73. By contrast, vote feedback did affect whether or not *ss* individuals chose to participate on the honor committee – 92% of those voted leader chose to participate on the honor committee, compared to only 43% of those voted weakest, $\chi^2$ (1) = 6.75, $p$ = .01. (Refer to Figure 4.)

I also ran these analyses using background questionnaire scores as covariates, and the Condition x Genotype interaction was always significant (i.e., all $p$'s < .05.). Additionally, I conducted binary logistic regressions using time 2 hormone levels and change in hormone levels, but neither the hormone levels nor their interactions with vote feedback and genotype showed significant effects.

Figure 3: Predicted probabilities of choosing to participate on the honor committee as a function of genotype, vote feedback, and baseline testosterone.

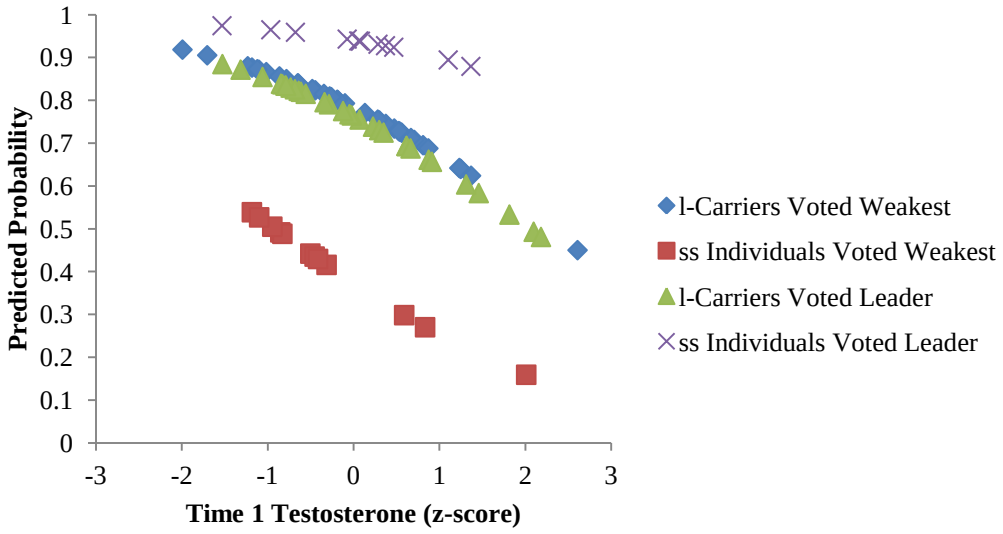
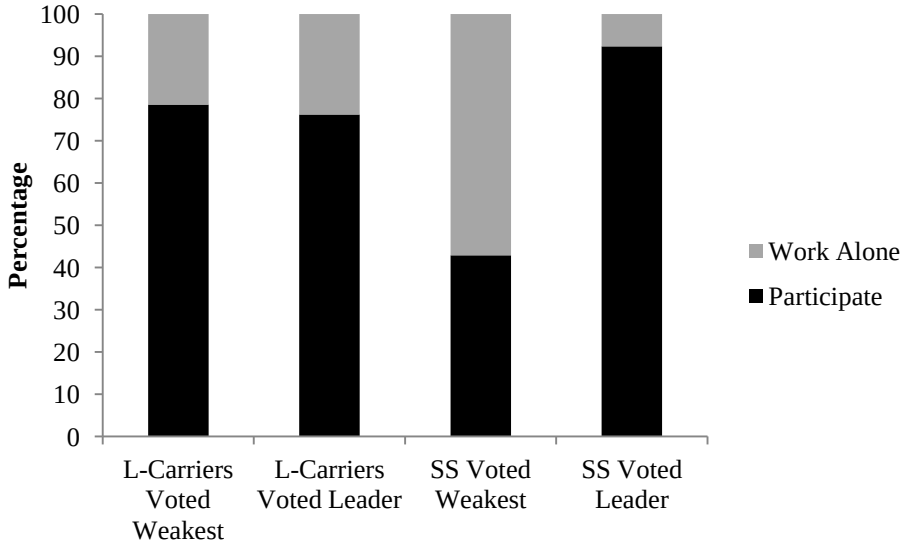



Figure 4: Percentage of participants who decided to participate or work alone as a function of genotype and vote feedback.

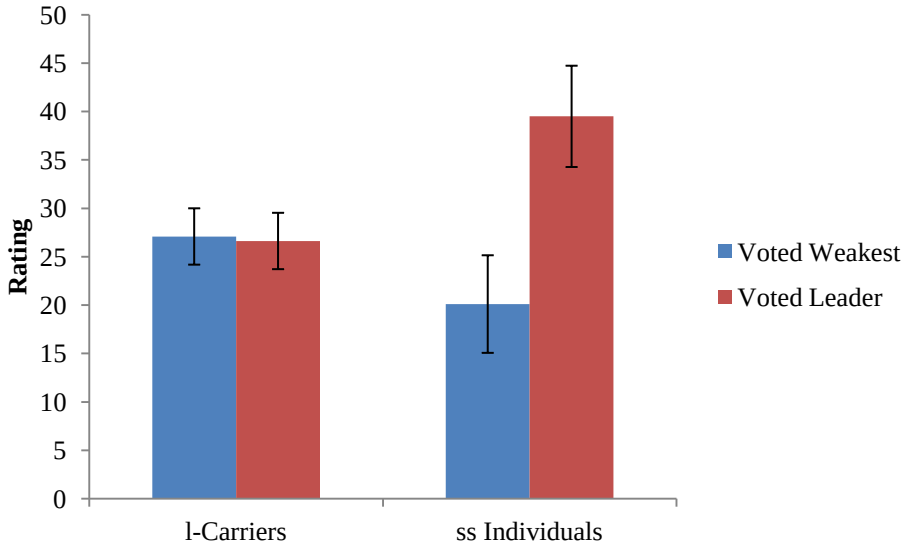


**Dating Anxiety**

It was predicted that after being voted leader, *ss* individuals would be less anxious about approaching a potential mate, but after being voted weakest, *ss* individuals would be more anxious. Hormones were not predicted to moderate responses to the dating anxiety questionnaire. Nevertheless, I conducted preliminary regression analyses using Time 1 C and Time 1 T, in addition to Condition and Genotype, as predictors. These analyses either revealed no significant effects of baseline hormones or, if significant, failed OLS regression assumptions. Regression analyses incorporating time 2 hormone levels, as well as both baseline and time 2 testosterone-to-cortisol ratios, also did not reveal any significant effects of hormones on responses to any of the seven dating questions. Therefore, responses to questions on the dating anxiety task were entered into separate 2 (Condition: voted leader vs. voted weakest) x 2 (Genotype: *l*-carriers vs. *ss* individuals) x 2 (Attractiveness: attractive vs. unattractive) mixed-model ANOVAs, with Condition and Genotype as the between-subjects variables and Attractiveness as the within-subjects variable. Because these analyses did not include hormonal measurements, the data from all participants were included.

Contrary to what was predicted, there were no gene-environment effects on anxiety; however, for the question: "If she did say 'no,' how much would that bother or upset you?" there was a significant Condition x Genotype interaction, $F(1, 107) = 5.65$, $p = .02$, partial $\eta^2 = .050$. As shown in Figure 5, regardless of whether the woman was attractive or unattractive, *ss* individuals voted leader said they would be more upset than *l*-carriers, while *ss* individuals voted weakest would be less upset than *l*-carriers. This suggests that the vote feedback differentially affected *ss* individuals' sensitivity to rejection.

Figure 5: Ratings of how upset *l*-carriers and *ss* individuals would be if they were rejected after asking a woman out as a function of vote feedback.




**Minimum Acceptable Mate Selection Criteria**

It was predicted that after being voted leader, *ss* individuals would increase their minimum standards for traits like intelligence and emotional stability when considering a potential one-night stand, but after being voted weakest, *ss* individuals would lower their standards. Hormones were not predicted to moderate the responses to the minimum acceptable mate selection criteria questionnaire. Nevertheless, I conducted preliminary regression analyses using Time 1 C and Time 1 T, in addition to Condition and Genotype, as predictors. These analyses either revealed no significant effects of baseline hormones or, if significant, failed OLS regression assumptions. Regression analyses incorporating time 2 hormone levels, as well as both baseline and time 2 testosterone-to-cortisol ratios, also did not reveal any significant effects of hormones on minimum acceptable percentiles. Therefore, the minimum acceptable percentiles for the 14 mate

criteria were entered into separate 2 (Condition: voted leader vs. voted weakest) x 2 (Genotype: *l*-carriers vs. *ss* individuals) x 3 (Mate: one-night stand vs. date vs. marriage partner) mixed-model ANOVAs, with Condition and Genotype as the between-subjects variables and Mate as the within-subjects variable. Because these analyses did not include hormonal measurements, the data from all participants were included.

As expected, traits that are most important for male reproductive success (e.g., attractiveness), as well as traits that are least important (e.g., wealth, dominance), showed no significant differences in minimum acceptable percentiles as a function of vote feedback, genotype, and/or type of mate. There were, however, significant Condition x Genotype x Mate interactions for the traits of Intelligence: $F(2, 107) = 3.39$, $p = .04$, partial $\eta^2 = .031$; Emotional Stability: $F(2, 107) = 3.20$, $p = .04$, partial $\eta^2 = .029$; Friendliness, $F(2, 107) = 3.94$, $p = .02$, partial $\eta^2 = .036$; and Sense of Humor: $F(2, 107) = 3.19$, $p = .04$, partial $\eta^2 = .029$. Follow-up 2 (Condition) x 2 (Genotype) ANOVAs revealed that these effects are driven by *ss* individuals voted leader having higher minimum percentiles and *ss* individuals voted weakest having lower minimum percentiles for one-night stands (see Figures 6-9).

Figure 6: Minimum acceptable percentiles for intelligence for a marriage partner, a date, and a one-night stand as a function of genotype and vote feedback.

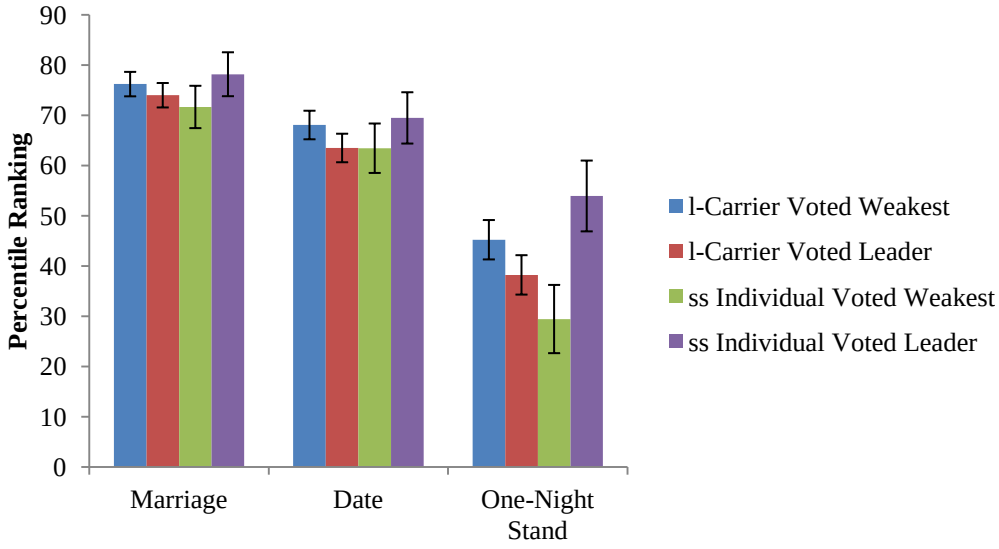



Figure 7: Minimum acceptable percentiles for emotional stability for a marriage partner, a date, and a one-night stand as a function of genotype and vote feedback.

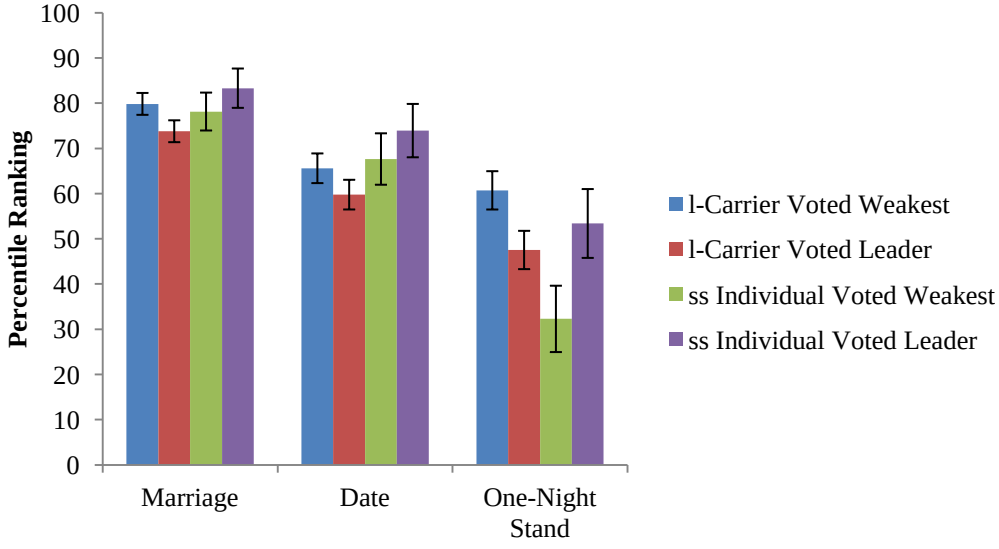


Figure 8: Minimum acceptable percentiles for friendliness for a marriage partner, a date, and a one-night stand as a function of genotype and vote feedback.

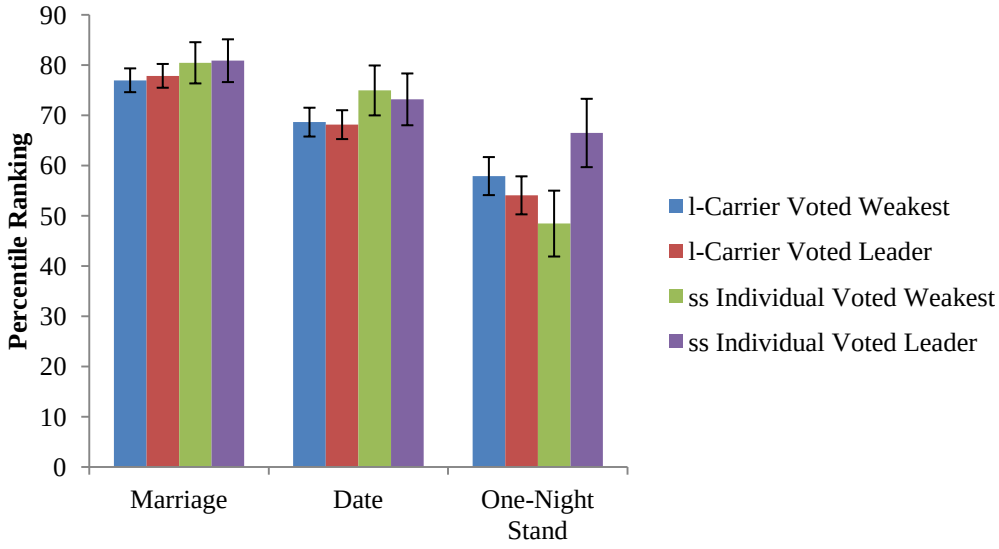
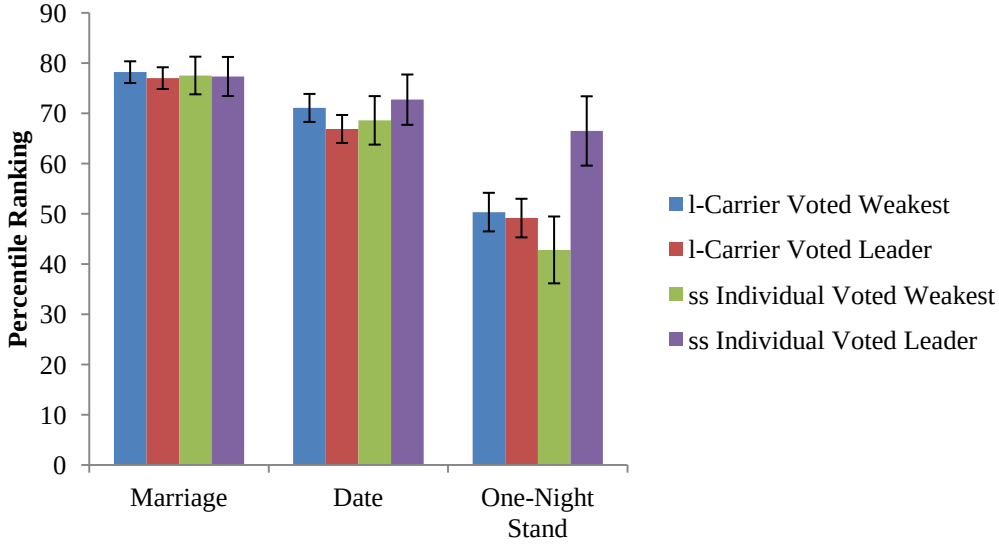



Figure 9: Minimum acceptable percentiles for sense of humor for a marriage partner, a date, and a one-night stand as a function of genotype and vote feedback.



There was also a significant Condition x Genotype interaction for Intelligence, $F(1, 107) = 5.84$, $p = .02$, partial $\eta^2 = .052$, demonstrating that *ss* individuals voted leader reported higher minimum percentiles and *ss* individuals voted weakest reported lower minimum percentiles for intelligence (refer to Figure 10). In addition, there was a significant Condition x Genotype interaction for Emotional Stability: $F(1, 107) = 6.23$, $p = .02$, partial $\eta^2 = .055$. As shown in Figure 11, while *ss* individuals voted leader reported higher minimum percentiles and *ss* individuals voted weakest reported lower minimum percentiles for emotional stability. Interestingly, the opposite was true of *l*-carriers.

Figure 10: Minimum acceptable percentiles for intelligence for a romantic partner as a function of genotype and vote feedback.

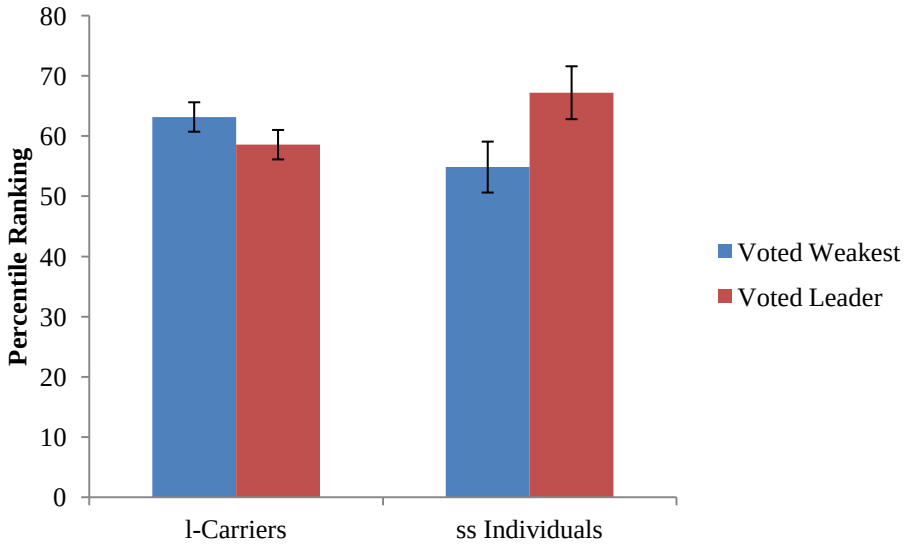
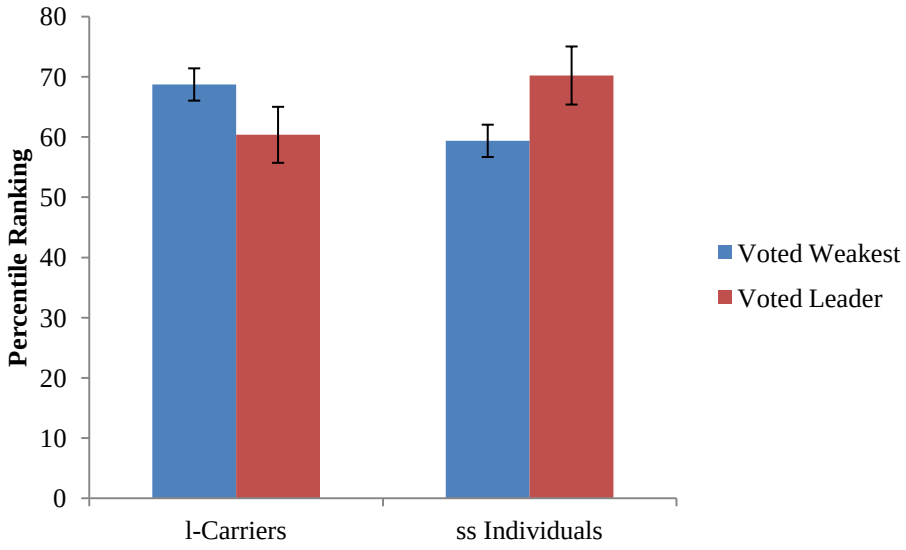


Figure 11: Minimum acceptable percentiles for emotional stability for a romantic partner as a function of genotype and vote feedback.




I also examined the correlations between self-ratings and minimum acceptable criteria for a marriage partner, date, and one-night stand. Kenrick et al. (1993) found that men's self-ratings tend to be correlated with their minimum acceptable criteria for marriage partners but not one-night stands. However, if *l*-carriers are not affected by social feedback, then their self-ratings should be related to their minimum acceptable criteria for all relationship types. By contrast, *ss* individuals' self-ratings should be correlated with their minimum acceptable criteria for a marriage partner and a date but not for a one-night stand. As shown in Tables 2-5, this appears to be the trend.

Table 2: Correlations between self-ratings and minimum acceptable intelligence for a marriage partner, date, and one-night stand.

| | **Intelligence** | | ***z*** |
|---|---|---|---|
| | ***ll, ls*** **(n = 84)** | ***ss*** **(n = 27)** | **(one-tailed)** |
| **Marriage Partner** | .573*** | .696*** | -.89 |
| **Date** | .540*** | .443** | .55 |
| **One-night Stand** | .187* | -.156 | 1.49* |

*$p < .10$ **$p < .05$, ***$p < .01$

Table 3: Correlations between self-ratings and minimum acceptable emotional stability for a marriage partner, date, and one-night stand.

| | **Emotional Stability** | | ***z*** |
|---|---|---|---|
| | ***ll, ls*** **(n = 84)** | ***ss*** **(n = 27)** | **(one-tailed)** |
| **Marriage Partner** | .508*** | .481** | .15 |
| **Date** | .440*** | .259 | .89 |
| **One-night Stand** | .228** | .034 | .85 |

*$p < .10$ **$p < .05$, ***$p < .01$

Table 4: Correlations between self-ratings and minimum acceptable friendliness for a marriage partner, date, and one-night stand.

| | **Friendliness** | | ***z*** |
|---|---|---|---|
| | ***ll, ls*** **(n = 84)** | ***ss*** **(n = 27)** | **(one-tailed)** |
| **Marriage Partner** | .643*** | .610*** | .23 |
| **Date** | .519*** | .441** | .44 |
| **One-night Stand** | .415*** | .083 | 1.54* |

*$p < .10$ **$p < .05$, ***$p < .01$

Table 5: Correlations between self-ratings and minimum acceptable sense of humor for a marriage partner, date, and one-night stand.

| | **Sense of Humor** | | ***z*** |
|---|---|---|---|
| | ***ll, ls*** **(n = 84)** | ***ss*** **(n = 27)** | **(one-tailed)** |
| **Marriage Partner** | .667*** | .665*** | .02 |
| **Date** | .515*** | .536*** | -.12 |
| **One-night Stand** | .194* | -.012 | .90 |

*$p < .10$ **$p < .05$, ***$p < .01$

# Chapter V: Discussion

The results of the present study support the differential susceptibility hypothesis. First, in order to demonstrate differential susceptibility, genotype (i.e., the susceptibility factor) needed to be independent of both the environmental factor and the outcome variables (Belsky et al., 2007). This criterion was met. By design, social status threat or confirmation (i.e., the environmental factor) was independent of genotype. Then, because there were no significant main effects of genotype, it can be concluded that genotype was not independently associated with any of the outcome variables.

Next, there needed to be a cross-over interaction (Belsky & Pluess, 2009), and this was demonstrated in men's testosterone responses. As predicted, *l*-carriers' were not only less hormonally reactive but their testosterone levels were also not affected by the vote feedback. By contrast, low cortisol (approach) *ss* individuals increased in testosterone after being voted into the highest status position and decreased in testosterone when voted into the lowest status position. High cortisol (avoidant) *ss* individuals decreased in testosterone after being voted into the highest status position but increased in testosterone when voted into the lowest status position. Thus, *ss* individuals respond "negatively" to being placed in positions that do not match their baseline levels of behavioral approach; that is, *ss* men with high baseline levels of approach behavior find it more stressful to be placed in the low status position, while *ss* men with high baseline levels of avoidant behavior find it more stressful to be in the high status position. Conversely, increases in testosterone could be interpreted as *ss* individuals responding "positively" to being placed in status positions that match their baseline levels of behavioral approach.

Finally, in further support and extension of the differential susceptibility hypothesis, the *s* allele was associated with adaptive behavioral plasticity. That is, while *l*-carriers were not affected by vote feedback when deciding whether or not to participate on the honor committee, *ss* individuals were less likely to participate after being voted weakest and more likely to participate after being voted leader. Moreover, both of these responses reflect adaptive strategies. On the one hand, readily assuming the role of leader when that role is conferred by one's peers enables one to gain status without incurring the costs of directly competing for status (e.g., the risk of physical harm). On the other hand, withdrawing from the group when the consensus appears to be that one's status is lower than that of the other group members could be viewed as a strategy for avoiding conflict or additional threats, which would have been particularly adaptive for susceptible individuals, who are more physiologically sensitive to stress (Belsky & Pluess, 2009).

Because participants' decisions to participate on the committee or work alone were made 20 minutes after the vote feedback manipulation, and about 40 minutes after the collection of baseline measurements, the influence of hormone levels on this decision may have been attenuated by the time elapsed. Accordingly, I did not find a significant effect of cortisol or any significant interactions between hormone levels and either genotype or condition affecting participants' decisions. Nevertheless, there was an unpredicted main effect of testosterone, such that lower baseline testosterone predicted a higher probability of choosing to work with on the committee. This may reflect the fact that lower testosterone is associated with more cooperative behavior. For instance, Mehta et al. (2009) found that men and women with low testosterone performed better when they had to work as a team to outcompete another group, while men and women with high testosterone performed better when they worked alone to outcompete a

competitor. Thus, higher testosterone individuals in this study may not have been as motivated to engage in a cooperative activity.

The *s* allele was also associated with conditional shifts in mating psychology. In general, *l*-carriers' mating psychology did not change as a function of vote feedback. For *ss* individuals, on the other hand, two important trends emerged. First, although the original prediction that vote feedback would differentially affect *ss* individuals' dating anxiety was not supported, I did find that being voted leader increased sensitivity to rejection in *ss* individuals, whereas being voted weakest decreased sensitivity. Nevertheless, this response still makes evolutionary sense: women prefer high status men (Buss, 1989), so high status me are less likely to be rejected, and a rejection could be perceived as a status threat. Therefore, it is adaptive for *ss* men with high status to be more vigilant for and sensitive to rejection by potential mates. Furthermore, the fact that the predicted gene-environment interaction effect on dating anxiety was not supported is not necessarily evidence against differential susceptibility; instead, it may actually support recent research that suggests the *s* allele is more closely related to "social sensitivity," as opposed to general anxiety or neuroticism (Canli & Lesch, 2007; Homberg & Lesch, 2011).

Second, for one-night stands, *ss* individuals lowered their standards for intelligence, emotional stability, friendliness, and sense of humor after being voted weakest but raised their standards after being voted leader. Thus, it appears that *ss* men are matching their standards to their current status, whereas *l*-carriers are not. This sort of shift would have been adaptive insofar as it increased mating opportunities or at least ensured that the number of mating opportunities one encountered did not vary. Moreover, matching standards to one's status would reduce the likelihood of rejection (Regan, 1998b), which again may be particularly beneficial for susceptible individuals.

## STRENGTHS AND LIMITATIONS

One strength of this study is its approach to testing differential susceptibility. Examining the effects of specific environmental factors (or adaptive problems) on outcome variables – endocrine response, dominance/affiliation, and mating – that are likely more directly affected by genetic differences in serotonergic function (Kiser, Steemers, Branchi, & Homberg, 2012) enables us to make more specific predictions about the nature of gene-environment interactions. The strength of this approach is further supported by a recent meta-analysis, demonstrating that studies examining specific stressors (e.g., childhood maltreatment) found a significant association between the *s*-allele, stress, and the development of depression, whereas those that examined more general measures of stress (e.g., stressful life events) did not (Karg, Burmeister, Shedden, & Sen, 2011). In addition, this study adds to evidence from past research, suggesting that the contexts in which one should observe differential susceptibility associated with the 5-HTTLPR are social in nature (Homberg & Lesch, 2011; Verschoor & Markus, in press). Still, this study did not include a direct test of non-social environmental factors (or adaptive problems), such as food scarcity/abundance or animal threats, as opposed to emotional or social threats; therefore, it may be beneficial for future research to compare non-social and social adaptive problems to determine if one or the other has a greater effect.

Next, although the honor committee paradigm used in this study was successful in eliciting differential responses in *ss* individuals, it may still have some limitations. For instance, the vote feedback may confound social status and social exclusion because being voted weakest and out of the committee meant one was not only low status but, also, excluded from the committee. Thus, *ss* men may have been reacting to the low status position, the social exclusion, or the combination of the two. In addition, many

past endocrine studies used competitive manipulations, which clearly assess dominance. However, in the present study, when the honor committee was described to participants, it was explained that students are elected to honor committees on the basis of their "character." Moreover, by having participants vote, status was (perceived to be) conferred, not won. This suggests that paradigm used in this study may have had less to do with dominance and more to do with prestige because, whereas a dominant individual achieves status through direct competition and aggression, status is conferred to a prestigious individual by his peers because of his character – his knowledge, skills, and *leadership* ability (Henrich & Gil-White, 2001). Nevertheless, both dominance and prestige confer similar benefits, such as more mating opportunities and greater social influence (von Rueden, Gurven, & Kaplan, 2011); therefore, even if the honor committee actually represents a prestige hierarchy, the effects of gaining or losing prestige should be similar to the effects observed in past endocrine research of gaining or losing dominance. Moreover, if the honor committee paradigm is more closely related to prestige, it may actually reflect how men respond in "natural" social groups. For instance, when McIntyre, Li, Chapman, Lipson, and Ellison (2011) did not find any relationship between dominance and social status among college men, they concluded that it may be due to status being conferred rather than won through direct competition.

Another possible limitation is that, although participants did provide self-ratings for the mate criteria, this study did not include a direct measure of mate value. There is currently no empirically validated scale for measuring mate value, but it may still be important to assess self-perceived mate value in future studies because it could be differentially affected by vote feedback and/or moderate changes in mating psychology (e.g., Penke & Denissen, 2008). Then again, Regan (1998a, 1998b) found that self-perceived mate value did not correlate with men's minimum acceptable criteria, which

suggests mate value may not have been be related to men's responses to the minimum acceptable criteria questionnaire in the present study.

There were also were no differences between *l*-carriers and *ss* individuals on any of online self-report measures, which is somewhat surprising because, given that *s*-allele is associated with greater sensitivity, one might expect genetic differences for scales like the Highly Sensitive Person Scale, especially the subscales of Ease of Excitability and Low Sensory Threshold. However, participants completed questionnaires online at their convenience. As a result, I had very little control over how seriously participants considered their responses, and it is possible that their scores are not accurate. Moreover, self-reports tend to be inaccurate in general due to factors such as poor scale construction, response-biases, and lack of self-insight (Mehl & Pennebaker, 2003; Nisbett & Wilson, 1977; Wilson & Nisbett, 1978).

Finally, another limitation of this study is that it included only male participants, so the results may not generalize to women. Then again, from an evolutionary perspective, social status was more important to men's fitness; therefore, the paradigm used in this study may not be as evolutionarily salient to women. Furthermore, measuring dating anxiety and minimum acceptable mate criteria would not be appropriate for assessing changes in women's mating psychology because women are generally less likely to approach and ask out potential mates (e.g., Impett & Peplau, 2003; McNamara & Grossman, 1991; Rose & Frieze, 1993), and they are more selective regardless of the type of relationship (Kenrick et al., 1990, 1993). Thus, in future studies, researchers may want to consider sex differences in the types of adaptive problems that susceptible individuals are differentially responsive to and the types of conditional responses susceptible individuals should exhibit. For instance, whereas a man's status affects his ability to attract mates, a woman's attractiveness has important implications for the types

of mates she can attract because men prioritize physical attractiveness when selecting mates (Buss, 1994). Therefore, manipulating feedback about attractiveness or manipulating the attractiveness of a woman's peers could elicit different tactics of mate attraction and intrasexual competition in women carrying the *s*-allele. For example, Durante, Li, and Haselton (2008) found that women who perceive themselves as less attractive were more likely to wear more revealing clothing to attract a mate and take attention away from their more attractive rivals. Thus, susceptible women may show an increased preference for revealing clothing after receiving negative feedback about their attractiveness, whereas susceptible women who receive positive feedback may show a decreased preference for revealing clothing.

## CONCLUSION

The present study supports the differential susceptibility hypothesis and demonstrates the need for a more evolutionary approach to understanding the physiological, behavioral, and psychological characteristics of those with susceptible genotypes. We should not expect the *s* allele to be related to all possible "negative" outcomes in response to all types of negative environmental stimuli, nor should we necessarily expect the *s* allele to be associated with any given mental disorder, as mental disorders encompass multiple symptoms that are not always evolutionarily related in terms of cause and/or function (see Wakefield, 2005). Instead, the *s* allele should be associated with specific responses (e.g., social withdrawal) to specific environmental factors (e.g., status loss or social exclusion) that function to reduce social conflict and prevent further stress system activation. Similarly, we should also not expect the *s* allele to be related to all possible "positive" outcomes in response to all types of positive environments. Rather, the *s* allele should be associated with specific responses (e.g.,

dominance and/or affiliative behavior) in response to specific environmental factors (e.g., conferral of social status or support) that function to solidify status or social bonds and maintain low stress system activation.

Ultimately, this new perspective has implications for other aspects of susceptible individuals' psychology. For instance, Chiao (2010) suggests that *s*-carriers should have a preference for hierarchical social structures because hierarchies create certainty in social interaction, which may explain why the *s* allele is more common in collectivistic cultures, which endorse more rigid social hierarchies (Chiao & Blizinsky, 2010). But, instead of relying on broad measures of individualism and collectivism, the need for certainty in social interactions could be tested in terms of an individual's sensitivity to social norm violations and conformity, as well as his/her own tendencies toward conformity or nonconformity. Social norm violations could threaten hierarchy stability or signal that a person may be an unreliable social exchange partner, whereas conformity signals the opposite (Wenegrat, Abrams, Castillo-Yee, & Romine, 1996; Wenegrat, Castillo-Yee, & Abrams, 1996). Thus, are *ss* individuals quicker to detect social norm violations, as well as social norm compliance (i.e., conformity)? If so, are they differentially responsive (e.g., becoming less cooperative with a person who violates norms and more cooperative with a person conforms)? Given recent research suggesting that the *l* allele may be associated with psychopathy (Glenn, 2011), which in turn, is related to deficits in the ability to detect social norm violations (Ermer & Kiehl, 2010), this may prove to be a fruitful direction for future research.

# Appendices

## APPENDIX A: DEMOGRAPHICS SURVEY

**What is your birthday?**
mm_____ dd_____ yyyy_____

**Sex:**
_____ Male
_____ Female

**What is your height?**
_____ Feet _____ Inches

**What is your ethnicity? (Select one)**
_____ African American/African/Black
_____ Asian American/Asian
_____ Caucasian/White
_____ Hispanic
_____ Middle Eastern
_____ Native American
_____ South Asian (e.g. Indian, Pakistani)
_____ Other

**What is your family's socioeconomic status? (Select one)**
_____ Lower class
_____ Lower-middle class
_____ Middle class
_____ Upper-middle class
_____ Upper class

**Which sex are you attracted to? (Select one)**
_____ Men
_____ Women
_____ Both

**Are you currently in a committed, romantic relationship? (Select one)**
_____ Yes
_____ No

## APPENDIX B: DATING ANXIETY QUESTIONNAIRE

***Imagine asking the woman in the photo to meet you or to go on a date with you. Use the 0-100 scale below to respond to the following questions.***

0 = Not at all (Extremely low)
50 = Moderately (Moderate)
100 = Completely (Extremely high)

1. Ignoring whether or not she'd be interested in you, how interested are you in meeting her?

_____

2. How likely would you be to actually ask her to meet you (to go out with you)?

_____

3. Realistically, how anxious or nervous would you be to ask her to meet you (to go out with you)?

_____

4. How interested do you think she would be?

_____

5. How likely is she to say "no"?

_____

6. If she did say "no," how much would that bother or upset you?

_____

7. How likely would you be to meet (go out with) her if *she asked you*?

_____

## APPENDIX C: MATE SELECTION CRITERIA QUESTIONNAIRE

***For this part of the survey, you will be using percentile ranking to describe the characteristics pertaining to your ideal romantic partners. The percentiles correspond to how a person stacks up against all the people you might encounter on the street or a college campus during a typical week. To make sure you understand percentile scales, let's go through an example. Suppose you are a male and that your relevant population of potential mates is women. So, consider the characteristic of friendliness. If we could rank all the women by their friendliness, then the friendliest woman would be at the 100$^{th}$ percentile of friendliness – she is friendlier than 100% of all women. The most <u>un</u>friendly woman is at the 0$^{th}$ percentile of friendliness – she is friendlier than 0% of all women. The woman at the 50$^{th}$ percentile of friendliness is friendlier than exactly 50% of all women and less friendly than 49% of the people on this dimension.***

For each of the following characteristics, give the **minimum** percentile that you would find acceptable in a partner ***for a date***.

**Percentile**

_____ Kindness/understanding
_____ Intelligence
_____ Earning capacity
_____ Physical attractiveness
_____ Aggressiveness
_____ Emotional stability
_____ Friendliness
_____ Popularity
_____ Sexiness
_____ Wealth
_____ Ambition
_____ Sense of humor
_____ Social status
_____ Dominance

For each of the following characteristics, give the **minimum** percentile that you would find acceptable in a partner ***for marriage***.

**Percentile**

_____ Kindness/understanding
_____ Intelligence
_____ Earning capacity
_____ Physical attractiveness
_____ Aggressiveness
_____ Emotional stability
_____ Friendliness
_____ Popularity
_____ Sexiness
_____ Wealth
_____ Ambition
_____ Sense of humor
_____ Social status
_____ Dominance

For each of the following characteristics, give the **minimum** percentile that you would find acceptable in a partner ***for a one-night stand***. (NOTE: The one-night stand would involve a person who you did not know previously and would not see again. Although you may not be inclined to engage in such relationships, make the best assessment that you can.)

**Percentile**

_____ Kindness/understanding
_____ Intelligence
_____ Earning capacity
_____ Physical attractiveness
_____ Aggressiveness
_____ Emotional stability
_____ Friendliness

_____ Popularity
_____ Sexiness
_____ Wealth
_____ Ambition
_____ Sense of humor
_____ Social status
_____ Dominance

***For this part of the survey, you will be using percentile rankings to describe the characteristics pertaining to <u>yourself</u>. The percentiles correspond to how you stack up against all the men you might encounter on the street or a college campus during a typical week.***

Indicate your percentile ranking on each of the following characteristics.

**Percentile**

_____ Kindness/understanding
_____ Intelligence
_____ Earning capacity
_____ Physical attractiveness
_____ Aggressiveness
_____ Emotional stability
_____ Friendliness
_____ Popularity
_____ Sexiness
_____ Wealth
_____ Ambition
_____ Sense of humor
_____ Social status
_____ Dominance

## APPENDIX D: EXPERIMENT SCRIPT

OK. Now, I would like to discuss the rest of the study. This study is part of a larger initiative to create a student Honor Committee that will preside over cases of academic dishonesty, such as cheating and plagiarism, and other violations of the honor code. As it now stands, University staff members are responsible for disciplinary action (and I quote):

> "As authorized by the Board of Regents of The University of Texas System, the Office of the Dean of Students is responsible for the administration of student discipline…[The Dean's Office] routinely receives reports of alleged violations of University rules from campus offices, as well as individual faculty members and students. In response to such a report, [the Dean] generally schedules a meeting with the student to discuss the suspected violation(s)."

However, many other colleges and universities across the nation have or are considering student-administered honor systems. At these institutions, students are elected to serve on the Honor Committee by their peers on the basis of their character. Honor committee members are then responsible for investigating allegations of honor violations, holding hearings, and rendering verdicts.

The University of Texas Honor Code states:

> "The core values of the University of Texas at Austin are learning, discovery, freedom, leadership, individual opportunity, and responsibility. Each member of the University is expected to uphold these values through integrity, honesty, trust, fairness and respect towards peers and community."

Because the essence of the University's honor system is individual responsibility in all matters relating to a student's honor, *its students* should be entrusted to maintain the honor code and pass judgment on those who violate the code.

You are participating in a pilot program; the data from this program will ultimately be used in the formation of future student Honor Committees. At this stage, we are experimenting with group dynamics and basic personality processes that will yield the most effective committees. This is why the University has asked for help from experimental psychologists.

As a member of the Honor Committee, you would be responsible for determining guilt and administering punishments for fellow students who have been accused of scholastic dishonesty or misconduct. You would even have the authority to refer a student to the Office of Student Affairs with a recommendation for expulsion. It is a highly influential position that requires both leadership and integrity.

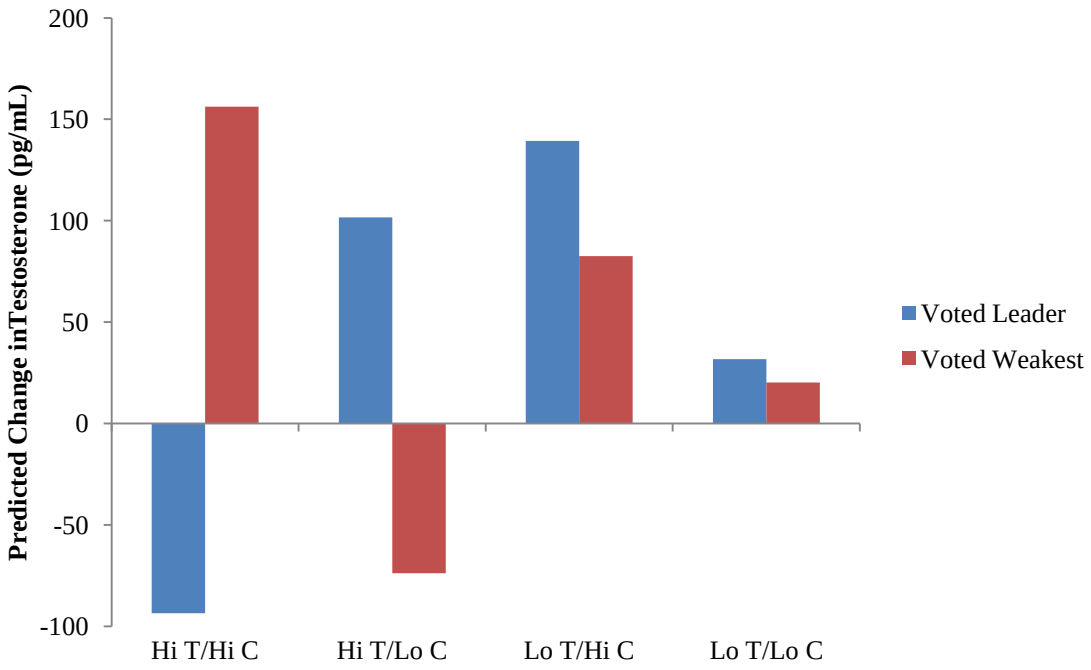
The Honor Committee consists of a team of individuals working closely together, guided by a strong leader. Based on the interaction you've just completed, we want you to nominate the one person in the group who you think has the strongest and best set of leadership skills, and who would make the best leader for an Honor Committee. Also, we want you to nominate the one person you think is the weakest, and therefore should not be on an Honor Committee. Please be aware that these are relative judgments – you may decide that no one is weak, but please nominate the weakest member of the group as the one who you think should not serve on an Honor Committee.

## APPENDIX E: SUPPLEMENTAL HORMONE DATA

Because past research has shown that the interaction between baseline testosterone and baseline cortisol predicts change in testosterone (Mehta & Josephs, 2010), I explored the possibility of an interaction between baseline testosterone and baseline cortisol predicting testosterone response. Thus, baseline testosterone (Time 1 T), baseline cortisol (Time 1 C), Condition (voted leader vs. voted weakest), Genotype (*l*-carriers vs. *ss* individuals), and their interactions were entered as predictors in a linear regression model predicting time 2 testosterone. Although the model was significant, $R^2 = .55$, $F(15, 75) = 5.99$, $p < .001$, the residuals were not normally distributed, so the model was re-run using log-transformed time 2 testosterone as the dependent variable.

The overall model was still significant, $R^2 = .53$, $F(15, 75) = 5.70$, $p < .001$, and there was a significant Time 1 T x Time 1 C x Condition x Genotype interaction, $\beta = -.056$, $p = .04$. Differential testosterone responses were only observed in *ss* individuals. I used the regression model to calculate predicted change in testosterone for *ss* individuals (refer to Figure 1E) in leader and weakest conditions at high and low baseline testosterone (i.e., mean baseline testosterone +/- 1 standard deviation) and at high and low baseline cortisol (i.e., mean baseline cortisol +/- 1 standard deviation). First, for *ss* individuals with high baseline testosterone, high baseline cortisol was associated with a decrease in testosterone when voted leader but an increase in testosterone when voted weakest, while low baseline cortisol was associated with an increase in testosterone when voted leader and decrease in testosterone when voted weakest. Next, *ss* individuals with low baseline testosterone tended to increase in testosterone, regardless of vote feedback; however, having higher baseline cortisol was associated with greater increases in testosterone. Ultimately, this suggests that the cross-over effect observed for testosterone response may be specific to *ss* individuals with high baseline testosterone.

Figure 1E: Predicted change in testosterone for *ss* individuals as a function of vote feedback and baseline testosterone and cortisol.

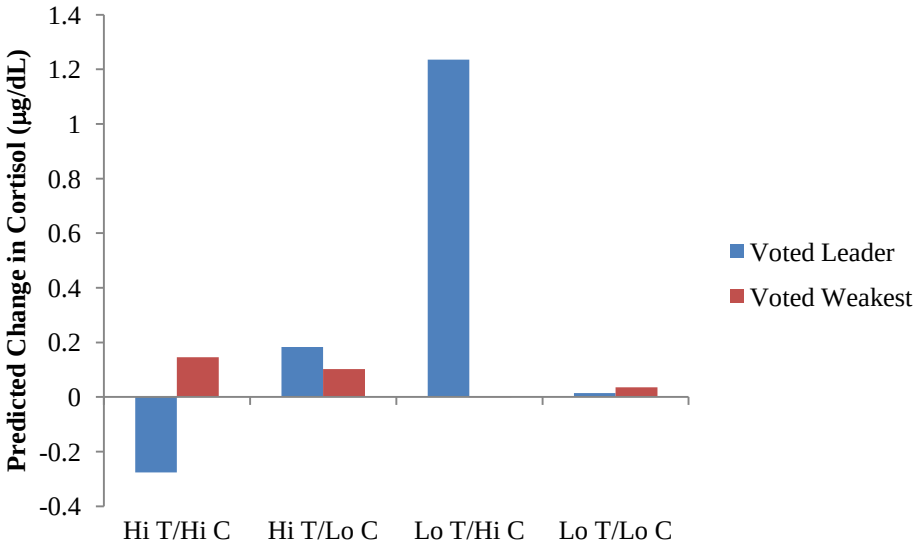



At first glance, it may seem counter-intuitive for low baseline testosterone individuals to increase in testosterone, especially when there is a status mismatch. However, cortisol is also differentially responsive to status, increasing under status mismatches conditions (i.e., stress) and decreasing under status match (i.e., non-stress) conditions (e.g., Josephs et al., in press; Mehta et al., 2008). Thus, to explore the possibility of an interaction between baseline testosterone and baseline cortisol predicting time 2 cortisol, baseline testosterone (Time 1 T), baseline cortisol (Time 1 C), Condition

(voted leader vs. voted weakest), Genotype (*l*-carriers vs. *ss* individuals), and their interactions were entered as predictors in a linear regression model predicting time 2 cortisol. However, because the residuals were not normally distributed, the model was re-run using log-transformed time 2 cortisol as the dependent variable.

The overall model was significant, $R^2 = .52$, $F(15, 75) = 5.42$, $p < .001$, and there was a significant Time 1 T x Time 1 C x Condition x Genotype interaction, $\beta = -.10$, $p = .05$. Importantly, this effect appears to be driven by differential cortisol responses in *ss* individuals with low baseline testosterone and high baseline cortisol. As shown in Figure 2E, I used the regression model to calculate predicted change in cortisol for *ss* individuals in leader and weakest conditions at high and low baseline testosterone (i.e., mean baseline testosterone +/- 1standard deviation) and at high and low baseline cortisol (i.e., mean baseline cortisol +/- 1standard deviation). Although low baseline testosterone/high baseline cortisol *ss* individuals did increase in testosterone regardless of vote feedback, they showed a large increase in cortisol after being voted leader but almost no change in cortisol after being voted weakest.

Figure 2E: Predicted change in cortisol for *ss* individuals as a function of vote feedback and baseline testosterone and cortisol.



# References

Aakvaag, A., Bentdal, O., Quigstad, K., Walstad, P., Ronningen, H., & Fonnum, F. (1978). Testosterone and testosterone binding globulin (TeBG) in young men during prolonged stress. *International Journal of Andrology, 1(1-6),* 22-31.

Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology, 12,* 169-177.

Alvergne, A., Faurie, C., & Raymond, M. (2009). Variation in testosterone levels and male reproductive effort: Insight from a polygynous human population. *Hormones and Behavior, 56(5),* 491-497.

Apicella, C.L., Dreber, A., Campbell, B., Gray, P.B., Hoffman, M., & Little, A.C. (2008). Testosterone and financial risk preferences. *Evolution and Human Behavior, 29,* 384-390.

Archer, J. (1991). The influence of testosterone on human aggression. *British Journal of Psychology, 82,* 1-28.

Aron, E., & Aron, A. (1997). Sensory-processing sensitivity and its relation to introversion and emotionality. *Journal of Personality and Social Psychology, 73,* 345-368.

Bailey, J.M., Kirk, K.M., Zhu, G., Dunne, M.P., & Martin, N.G. (2000). Do individual differences in sociosexuality represent genetic or environmentally contingent strategies? Evidence from the Australian Twin Registry. *Journal of Personality and Social Psychology, 78(2),* 537-545.

Bateup, H.S., Booth, A., Shirtcliff, E.A., & Granger, D.A. (2002). Testosterone, cortisol, and women's competition. *Evolution and Human Behavior, 23,* 181-192.

Baumeister, R.S., & Leary, M.R. (1995). The need to belong: Desire for interpersonal

attachments as a fundamental human motivation. *Psychological Bulletin, 117,* 497-529.

Beevers, C. G., Gibb, B. E., McGeary, J. E., & Miller, I. W. (2007). Serotonin transporter genetic variation and biased attention for emotional word stimuli among psychiatric inpatients. *Journal of Abnormal Psychology, 116*, 208 - 212.

Beevers, C.G., Wells, T.T., Ellis, A.J., & McGeary, J.E. (2009). Association of the serotonin transporter gene promoter region (5-HTTLPR) polymorphism with biased attention for emotional stimuli. *Journal of Abnormal Psychology, 118,* 670-681.

Belsky, J., Bakermans-Kranenburg, M.J., & van Ijzendoorn, M.H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science, 16,* 300-304.

Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B., & Williams, R. (2009). Vulnerability genes or plasticity genes? *Molecular Psychiatry, 14,* 746-754.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin, 135,* 885-098.

Benjet, C., Thompson, R.J., & Gotlib, I.H. (2010). 5-HTTLPR moderates the effect of relational peer victimization on depressive symptoms in adolescent girls. *Journal of Child Psychology and Psychiatry, 51,* 173-179.

Benham, G. (2006). The highly sensitive person: Stress and physical symptom reports. *Personality and Individual Differences, 40(7),* 1433-1440.

Berg, S.J., & Wynne-Edwards, K.E. (2001). Changes in testosterone, cortisol, estradiol levels in men becoming fathers. *Mayo Clinic Proceedings, 76,* 582-592.

Bogaert, A.F., & Fisher, W.A. (1995). Predictors of university men's number of sexual partners. *Journal of Sex Research, 32,* 119-130.

Bohnke, R., Bertsch, K., Kruk, M.R., & Naumann, E. (2010). The relationship between basal and acute HPA axis activity and aggressive behavior in adults. *Journal of Neural Transmission, 117,* 629-637.

Book, A.S., Starzyk, K.B., & Quinsey, V.L. (2001). The relationship between testosterone and aggression: a meta-analysis. *Aggression and Violent Behavior, 6,* 579-599.

Booth, A., & Dabbs, J.M. (1993). Testosterone and men's marriages. *Social Forces, 72,* 463-477.

Booth, A., Shelley, G., Mazur, A., Tharp, G., & Kittok, R. (1989). Testosterone, and winning and losing in human competition. *Hormones and Behavior, 23,* 556-571.

Boyce, W.T., & Ellis, B.J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17,* 271-301.

Brooks, J.H., & Reddon, J.R. (1996). Serum testosterone in violent and nonviolent young offenders. *Journal of Clinical Psychology, 52,* 472-483.

Brummett, B.H., Boyle, S.H., Siegler, I.C., Kuhn, C.M., Ashley-Koch, A., Jonassaint, C.R., …Williams, R.B. (2008). Effects of environmental stress and gender on associations among symptoms of depression and the serotonin transporter gene linked polymorphic region (5-HTTLPR). *Behavior Genetics, 38,* 34-43.

Burmeister, M., McInnis, M.G., & Zollner, S. (2008). Psychiatric genetics: progress amid controversy. *Nature Reviews Genetics, 9,* 527-540.

Burnham, T. (2007). High testosterone men reject low ultimatum game offers. *Proceedings of the Royal Society B, 274,* 2327-2330.

Burnham, T.C., Chapman, J.F., Gray, P.B., McIntyre, M.H., Lipson, S.F., & Ellison, P.T.

(2003). Men in committed, romantic relationships have lower testosterone. *Hormones and Behavior, 44,* 119-122.

Buss, A.H., & Perry, M.P. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology, 63,* 452-459.

Buss, D.M. (2009). How can evolutionary psychology successfully explain personality and individual differences? *Perspectives on Psychological Science, 4,* 359-366.

Buss, D.M. (1995). Evolutionary psychology: A new paradigm for psychological science. *Psychological Inquiry, 6,* 1-30.

Buss, D.M. (1994). The strategies of human mating. *American Scientist, 82,* 238-249.

Buss, D.M. (1989). Sex difference in human mate preferences: Evolutionary hypotheses tested in 37 cultures. *Behavioral and Brain Sciences, 12,* 1-49.

Buss, D.M. (1987). Sex differences in human mate selection criteria: An evolutionary perspective. In C. Crawford, M. Smith, & D. Krebs (Eds.), *Sociobiology and psychology: Ideas, issues, and applications* (pp. 335-351). Hillsdale, NJ: Erlbaum.

Buss, D.M., & Greiling, H. (1999). Adaptive individual differences. *Journal of Personality, 67,* 209-243.

Canli, T., & Lesch, K. (2007). Long story short: the serotonin transporter in emotion regulation and social cognition. *Nature Neuroscience, 10(9),* 1103-1109.

Carre, J.M., Muir, C., Belanger, J., & Putnam, S.K. (2006). Pre-competition hormonal and psychological levels of elite hockey players: Relationships to the 'home advantage.' *Physiology and Behavior, 89,* 392-398.

Carre, J.M., Putnam, S.K., & McCormick, C.M. (2009). Testosterone responses to competition predict future aggressive behavior at a cost to reward in men. *Psychoneuroendocrinology, 34,* 561-570.

Cashdan, E. (1995). Hormones, sex, and status in women. *Hormones and Behavior, 29,* 354-366.

Caspi, A., Sugden, K., Moffitt, T.E., Taylor, A., Craig, I.W., Harrington, H.L., …Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301,* 368-389.

Carstensen, H., Amer, B., Amer, I., & Wide, L. (1973). The postoperative decrease in plasma testosterone in men, after major surgery, in relation to FSH and LH. *Journal of Steroid Biochemistry, 4(1),* 45-55.

Chatterton, Jr., R.T., Vogelsong, K.M., Lu, Y., & Hudgens, G.A. (1997). Hormonal responses to psychological stress in men preparing for skydiving. *Journal of Clinical Endocrinology and Metabolism, 82(8),* 2503-2509.

Chiao, J.Y. (2010). Neural basis of social hierarchy across species. *Current Opinion in Neurobiology, 20(6),* 803-809.

Chiao, J.Y., & Blizinsky, K.D. (2010). Culture-gene coevolution of individualism-collectivism and the serotonin transporter gene. *Proceedings of the Royal Society B, 277,* 529-537.

Comings, D.E., Gade-Andavolu, R., Gonzalez, N., Wu, S., Muhleman, D., Blake, H., …MacMurray, J.F. (2000). A multivariate analysis of 59 candidate genes in personality traits: the temperament and character inventory. *Clinical Genetics, 58(5),* 375-385.

Cunningham, M.R. (1986). Measuring the physical in physical attractiveness: Quasi-experiments on the sociobiology of female facial beauty. *Journal of Personality and Social Psychology,* 50, 925-935.

Cunningham, M.R., Roberts, R., Barbee, A.P., Druen, P.B., & Wu, C. (1995). Their ideas of physical attractiveness are, on the whole, the same as ours: Consistency and

variability in the cross-cultural perception of female physical attractiveness. *Journal of Personality and Social Psychology, 68,* 261-279.

Dabbs, Jr., J.M., Carr, T.S., Frady, R.L., & Raid, J.K. (1995). Testosterone, crime, and misbehavior among 692 male prison inmates. *Personality and Individual Differences, 18,* 627-633.

Dabbs, Jr., J.M., Frady, R.L., Carr, T.S., & Besch, N.F. (1987). Saliva testosterone and criminal violence in young adult prison inmates. *Psychosomatic Medicine, 49,* 174-182.

Dabbs, Jr., J.M., & Haregrove, M.F. (1997). Age, testosterone, and behavior among female prison inmates. *Psychosomatic medicine, 59,* 447-480.

Dabbs, Jr., J.M., Jurkovic, G.J., & Frady, R.L. (1991). Salivary testosterone and cortisol among late adolescent male offenders. *Journal of Abnormal Child Psychology,* 19, 469-478.

Dabbs, Jr., J.M., Ruback, B., Frady, R.L., Hopper, C.H., & Sgoutas, D.S. (1988). Saliva testosterone and criminal violence among women. *Personality and Individual Differences, 9,* 269-275.

Dall, S.R.X., Houston, A.I., & McNamara, J.M. (2004). The behavioural ecology of personality: consistent individual differences from an adaptive perspective. *Ecology Letters, 7,* 734-739.

David, S.P., Murthy, N.V., Rabiner, E.A., Munafo, M.R., Johnstone, E.C., Jacob, R., …Grasby, P.M. (2005). A functional genetic variation of the serotonin (5-HT) transporter affects 5-HT1A receptor binding in humans. *Journal of Neuroscience, 25(10),* 2586-2590.

Decker, S.A. (2000). Salivary cortisol and social status among Dominican men. *Hormones and Behavior, 38,* 29-38.

DeWitt, T.J., Sih, A., & Wilson, D.S. (1998). Costs and limits of phenotypic plasticity. *Trends in Ecology and Evolution, 12,* 77-81.

Diver, M.J., Imitaz, K.E., Ahmad, A.M., Vora, J.P., & Fraser, W.D. (2003). Diurnal rhythms of serum total, free and bioavailable testosterone and of SHBG in middle-aged men compared with those in young men. *Clinical Endocrinology, 58(6),* 710-717.

Dunnlowski, U., Konrad, C., Kugel, H., Zwitserlood, P., Domschke, K., Schoning, S., …Suslow, T. (2010). Emotion specific modulation of automatic amygdala responses by 5-HTTLPR genotype. *NeuroImage, 53,* 893-898.

Durante, K.M., Li, N.P., & Haselton, M.G. (2008). Changes in women's choice of dress across the ovulatory cycle: Naturalistic and laboratory task-based evidence. *Personality and Social Psychology Bulletin, 34(11),* 1451-1460.

Eaves, L.J., Martin, N.G., Heath, A.C., Hewitt, J.K., & Neale, M.C. (1990). Personality and reproductive fitness. *Behavior Genetics, 20,* 563-568.

Ebstein, R. (2006). The molecular genetic architecture of human personality: Beyond self-report questionnaires. *Molecular Psychiatry, 11,* 427-445.

Edwards, D.A., & Kurlander, L.S. (2010). Women's intercollegiate volleyball and tennis: Effects of warm-up, competition, and practice on saliva levels of cortisol and testosterone. *Hormones and Behavior, 58,* 606-613.

Edwards, D.A., Wetzel, K., & Wyner, D.R. (2006). Intercollegiate soccer: Saliva cortisol and testosterone are elevated during competition, and testosterone is related to status and social connectedness with teammates. *Physiology & Behavior, 87,* 135-143.

Eley, T.C., Sudgen, K., Corsico, A., Gregory, A.M., Sham, P., McGuffin, P., …Craig, I.W. (2004). Gene-environment interaction analysis of serotonin system markers

with adolescent depression. *Molecular Psychiatry, 9,* 908-915.

Ellis, B.J., & Boyce, W.T. (2008). Biological sensitivity to context. *Psychological Science, 17,* 183-187.

Ellis, B.J., Essex, M.J., & Boyce, W.T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. *Development and Psychopathology, 17,* 303-328.

Ellis, B.J., Jackson, J.J., & Boyce, W.T. (2006). The stress response systems: Universality and adaptive individual differences. *Developmental Review, 26,* 175-212.

Ermer, E., & Kiehl, K.A. (2010). Psychopaths are impaired in social exchange and precautionary reasoning. *Psychological Science, 21(10),* 1399-1405.

Feinn, R., Nellissery, M., & Kranzler, H.R. (2005). Meta-analysis of the association of a functional serotonin transporter promoter polymorphism with alcohol dependence. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 133B,* 79-84.

Fisher, H.L., Cohen-Woods, S., Hosang, G.M., Uher, R., Powell-Smith, G., Keers, R., …McGuffin, P. (2012). Stressful life events and the serotonin transporter gene (5-HTT) in recurrent clinical depression. *Journal of Affective Disorder, 136,* 189-193.

Fleeson, W. (2001). Towards a structure- and process-integrated view of personality: Traits as density distributions of states. *Journal of Personality and Social Psychology, 80,* 1011-1027.

Fleming, A., Corter, C., Stallings, J., & Steiner, M. (2002). Testosterone and prolactin are associated with emotional responses to infant cries in new fathers. *Hormones and Behavior, 42,* 399-413.

Friedman, H.S., Tucker, J.S., Schwartz, J.E., Martin, L.R., Tomlinsonkeasey, C., Wingard, D.L., & Criqui, M.H. (1995). Childhood conscientiousness and longevity: Health behaviors and cause of death. *Journal of Personality and Social Psychology, 68,* 696-703.

Fuller, R.W. (1996). Serotonin receptors involved in regulation of pituitary-andrenocortical function in rats. *Behavioural Brain Research, 73,* 215-219.

Furmark, T., Tillfors, M., Garpenstrand, H., Marteinsdottir, I., Langstrom, B., Oreland, L., & Fredrikson, M. (2004). Serotonin transporter polymorphism related to amygdala excitability and symptom severity in patients with social phobia. *Neuroscience Letters, 362,* 189-192.

Gangestad, S.W., & Simpson, J.A. (2000). The evolution of human mating: Trade-offs and strategic pluralism. *Behavioral and Brain Sciences, 23,* 573-644.

Gelernter, J., Kranzler, H., & Cubells, J.F. (1997). Serotonin transporter protein (SCL6A4) allele and haplotype frequencies and linkage disequilibria in African- and European-American and Japanese populations and in alcohol-dependent subjects. *Human Genetics, 101,* 243-246.

Glenn, A.L. (2011). The other allele: Exploring the long allele of the serotonin transporter gene as a potential risk factor for psychopathy: A review of the parallels in findings. *Neuroscience and Biobehavioral Reviews, 35(3),* 612-620.

Gokturk, C., Schultze, S., Nilsson, K.W., von Knorring, L., Oreland, L., & Hallman, J. (2008). Serotonin transporter (5-HTTLPR) and monoamine oxidase (MAOA) promoter polymorphisms in women with severe alcoholism. *Archives of Women's Mental Health, 11,* 347-355.

Gotlib, I.H., Joormann, J., Minor, K.L., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression.

*Biological Psychiatry, 63,* 847-851.

Grammer, K. (1992). Variations on a theme: Age dependent mate selection in humans. *Behavioral and Brain Sciences, 15,* 100-102.

Grant, V.J., & France, J.T. (2001). Dominance and testosterone in women. *Biological Psychology, 58,* 41-47.

Grassi, L., Rossi, E., Cobianchi, M., Aguiari, L., Capozzo, M., Martinis, E., …Giraldi, T. (2010). Depression and serotonin transporter (5-HTTLPR) polymorphism in breast cancer patients. *Journal of Affective Disorders, 124(3),* 346-350.

Gray, P.B. (2003). Marriage, parenting, and testosterone variation among Kenyan Swahili men. *American Journal of Physical Anthropology, 122,* 279-286.

Gray, P.B., Kahlenberg, S.M., Barrett, E.S., Lipson, S.F., & Ellison, P.T. (2002). Marriage and fatherhood are associated with lower testosterone in males. *Evolution and Human Behavior, 23,* 193-201.

Gross, M.R. (1996). Alternative reproductive strategies and tactics: diversity within sexes. *Trends in Ecology and Evolution, 11(2),* 92-98.

Hallikainen, T., Saito, T., Lachman, H.M., Volavka, J., Pohjalainen, T., Ryynanen, O.P., …Tiihonen, J. (1999). Association between low activity serotonin transporter promoter genotype and early onset alcoholism with habitual impulsive violent behavior. *Molecular Psychiatry, 4,* 385-388.

Hariri, A.R., Drabant, E.M., Munoz, K.E., Kolachana, B.S., Mattay, V.S., Egan, M.F., & Weinberger, D.R. (2005). A susceptibility gene for affective disorders and the response of the human amygdala. *Archives of General Psychiatry, 62,* 146-152.

Hariri, A.R., & Holmes, A. (2006). Genetics of emotional regulation: the role of the serotonin transporter in neural function. *Trends in Cognitive Sciences, 10,* 182-191.

Hariri, A.R., Mattay, V.S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., …Weinberger, D.R. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297,* 400-403.

Harro, J., Merenakk, L., Nordquist, N., Konstabel, K., Comasco, E., & Oreland, L. (2009). Personality and the serotonin transporter gene: Associations in a longitudinal population-based study. *Biological Psychology, 81,* 9-13.

Heils, A., Teufel, A., Petri, S., Strober, G., Riederer, P., Bengel, D., & Lesch, K.P. (1996). Allelic variation of human serotonin transporter gene expression. *Journal of Neurochemisty, 66,* 2621-2624.

Henrich, J., & Gil-White, F.J. (2001). The evolution of prestige: Freely conferred deference as a mechanism for enhancing the benefits of cultural transmission. *Evolution and Human Behavior, 22,* 165-196.

Hermans, E.J., Putman, P., Baas, J.M., Koppeschaar, H.P., & van Honk, J. (2006). A single administration of testosterone reduces fear-potentiated startle in humans. *Biological Psychiatry, 59,* 872-874.

Hermans, E.J., Ramsey, N.F., & van Honk, J. (2008). Exogenous testosterone enhances responsiveness to social threat in the neural circuitry of social aggression in humans. *Biological Psychiatry, 63,* 263-270.

Hill, K., & Hurtado, A.M. (1996). *Ache life history: The ecology and demography of a foraging people*. New York: Aldine de Gruyter.

Hinckers, A.S., Laucht, M.L., Schmidt, M.H., Mann, K.F., Schumann, G., Schuckit, M.A., & Heinz, A. (2006). Low level of response to alcohol as associated with serotonin transporter genotype and high alcohol intake in adolescents. *Biological Psychiatry, 60,* 282-287.

Holmes, A., Murphy, D.L., & Crawley, J.N. (2003). Abnormal behavioral phenotypes of

serotonin transporter knockout mice: Parallels with human anxiety and depression. *Biological Psychiatry, 54,* 953-959.

Homberg, J.R., & Lesch, K. (2011). Looking on the bright side of serotonin transporter gene variation. *Biological Psychiatry, 69,* 513-519.

Hopcroft, R.L. (2006). Sex, status, and reproductive success in the contemporary United States. *Evolution and Human Behavior, 27(2),* 104-120.

Hucklebridge, F., Hussain, T., Evans, P., & Clow, A. (2005). The diurnal patterns of the adrenal steroids cortisol and dehydroepiandrosterone (DHEA) in relation to awakening. *Psychoneuroendocrinology, 30(1),* 51-57.

Hu, X., Oroszi, G., Chun, J., Smith, T.L., Goldman, D., & Schuckit, M.A. (2005). An expanded evaluation of the relationship of four alleles to the level of response to alcohol and the alcoholism risk. *Alcoholism: Clinical and Experimental Research, 29,* 8-16.

Impett, E., & Peplau, L.A. (2003). Sexual compliance: Gender, motivational, and relationship perspectives. *Journal of Sex Research, 40,* 87-100.

Jackson, D.N. (1984). *Personality research form manual (3rd ed.)*. Research Psychologists Press.

John, O.P., & Srivastava, S. (1999). The big-five trait taxonomy: History, measurement, and theoretical perspectives. In L.A. Pervin & O.P. John (Eds.), *Handbook of personality: Theory and research (2nd ed.)*, pp. 102-139, New York: Guildord Press.

Johnson, A.L., Gibb, B.E., & McGeary, J. (2010). Reports of childhood physical abuse, 5-HTTLPR, and women's attentional biases for angry faces. *Cognitive Therapy Research, 34,* 380-387.

Jones, A.C., & Josephs, R.A. (2006). Interspecies hormonal interactions between man

and the domestic dog (*Canis familiaris*). *Hormones and Behavior, 50,* 393-400.

Josephs, R. A., Newman, M. L, Brown, R. P., & Beer, J. M. (2003). Status, testosterone, and human intellectual performance: Stereotype threat as status concern. *Psychological Science, 14,* 158-163.

Josephs, R.A., Sellers, J.G., Newman, M.L., & Mehta, P.H. (2006). The mismatch effect: When testosterone and status are at odds. *Journal of Personality and Social Psychology, 90,* 999-1013.

Josephs, R.A., Telch, M.J., Hixon, J.M., Evans, J.J., Lee, H., Knopik, V.S., …Beevers, C.G. (in press). Genetic and hormonal sensitivity to threat: Testing serotonin transporter genotype x testosterone interaction. *Psychoneuroendocrinology*.

Kagan, J., Reznick, J., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58(6),* 1459-1473.

Kapuku, G.K., Treibner, F.A., & Davis, M.C. (2002). Relationships among socioeconomic status, stress-induced changes in cortisol, and blood pressure in African American males. *Annals of Behavioral Medicine, 24,* 320-325.

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited. *Archives of General Psychiatry, 68(5),* 444-454.

Keller, M.C., & Miller, G. (2006). Resolving the paradox of common, harmful, heritable mental disorders: Which evolutionary genetic models work best? *Behavioral and Brain Sciences, 29,* 385-452.

Kendler, K.S., Kuhn, J.W., Vittum, J., Prescott, C.A., & Riley, B. (2005). The interaction of stressful life events and a serotonin transporter polymorphism in the prediction of episodes of major depression. *Archives of General Psychiatry, 62,* 529-535.

Kenrick, D.T., Groth, G.E., Trost, M.R., & Sadalla, E.K. (1993). Integrating evolutionary

and social exchange perspectives on relationships: Effects of gender, self-appraisal, and involvement level on mate selection criteria. *Journal of Personality and Social Psychology, 64(6),* 951-969.

Kenrick, D.T., Sadalla, E.K., Groth, G., & Trost, M.R. (1990). Evolution, traits, and the stages of human courtship: Qualifying the parental investment model. *Journal of Personality, 58(1),* 97-116.

Kercher, A.J., Rapee, R.M., & Schniering, C.A. (2009). Neuroticism, life events and negative thoughts in the development of depression in adolescent girls. *Journal of Abnormal Child Psychology, 37,* 903-915.

Kidd, K.K. (2006). ALFRED – the Allele FREquency Database. http://alfred.med.yale.edu

Kiser, D., Steemers, B., Branchi, I., & Homberg, J.R. (2012). The reciprocal interaction between serotonin and social behaviour. *Neuroscience and Biobehavioral Reviews, 36,* 786-798.

Kruez, L.E., Rose, R.A., & Jennings, J.R. (1972). Suppression of plasma testosterone levels and psychological stress: A longitudinal study of young men in Officer Candidate School. *Archives of General Psychiatry, 26(5),* 479-482.

Kugeares, S.L. (2002). *Social anxiety in dating initiation an experimental investigation of an evolved mating-specific anxiety mechanism*. Unpublished doctoral dissertation, University of Texas at Austin, Austin, TX.

Kumakiri, C., Kodama, K., Shimizu, E., Yamanouchi, N., Okada, S., Noda, S., …Shirasawa, H. (1999). Study of the association between the serotonin transporter gene regulatory region polymorphism and personality traits in a Japanese population. *Neuroscience Letters, 263,* 205-207.

Kweon, Y.S., Lee, H.K., Lee, C.T., Lee, K., & Pae, C.U. (2005). Association of the

serotonin transporter gene polymorphism with Korean male alcoholics. *Journal of Psychiatric Research, 39,* 371-376.

Lakdawalla, Z., & Hankin, B.L. (2008). Personality as a prospective vulnerability to dysphoric symptoms among college students. *Journal of Psychopathology and Behavior Assessment, 30,* 121-131.

Laucht, M., Treutlen, J., Schmidt, B., Blomeyer, D., Becker, K., Buchmann, A.F., …Banaschewski, T. (2009). Impact of psychosocial adversity on alcohol intake in young adults: Moderation by the LL genotype of the serotonin transporter polymorphism. *Biological Psychiatry, 66,* 103-109.

Lee, Y.S., Choi, S.W., Han, D.H., Kim, D.J., & Joe, K.H. (2009). Clinical manifestation of alcohol withdrawal symptoms related to genetic polymorphisms of two serotonin receptors and serotonin trans porter. *European Addiction Research, 15,* 39-46.

Lesch, K., Bengel, D., Heils, A., Sabol, S.Z., Greenberg, B.D., Petri, S., …Murphy, D.L. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science, 274,* 1527-1532.

Levenson, M.R., Kiehl, K.A., & Fitzpatrick, C.M. (1995). Assessing psychopathic attributes in a noninstitutionalized population. *Journal of Personality and Social Psychology, 68,* 151-158.

Li, N.P., Bailey, J.M., Kenrick, D.T., & Linsenmeier, J.A.W. (2002). The necessities and luxuries of mate preferences: Testing the tradeoffs. *Journal of Personality and Social Psychology, 82(6),* 947-955.

Maurex, L., Zaboli, G., Ohman, A., Asberg, M., & Leopardi, R. (2010). The serotonin transporter gene polymorphism (5-HTTLPR) and affective symptoms among women diagnosed with borderline personality disorder. *European Psychiatry, 25,*

19-25.

Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral and Brain Sciences, 21,* 353-397.

Mazur, A., Booth, A., & Dabbs, Jr., J.M. (1992). Testosterone and chess competition. *Social Psychology Quarterly, 55(1),* 70-77.

McCaul, K.D., Gladue, B.A., & Joppa, M. (1992). Winning, losing, mood, and testosterone. *Hormones and Behavior, 26,* 486-504.

McHugh, R.K., Hofmann, S.G., Asnaani, A., Sawyer, A.T., & Otto, M.W. (2010). The serotonin transporter gene and risk for alcohol dependence: A meta-analytic review. *Drug and Alcohol Dependence,* 108, 1-6.

McIntyre, M.H., Li, A.Y., Chapman, J.F., Lipson, S.F., & Ellison, P.T. (2011). Social status, masculinity, and testosterone in young men. *Personality and Individual Differences, 51(4),* 392-396.

McNamara, J.R., & Grossman, K. (1991). Initiation of dates and anxiety among college men and women. *Psychological Reports, 69,* 252-254.

Mehl, M. R., & Pennebaker, J. W. (2003). The sounds of social life: A psychometric analysis of students' daily social environments and natural conversations. *Journal of Personality and Social Psychology, 84,* 857-870.

Mehrabian, A. (1994). Evidence bearing on the Affiliative Tendency (MAFF) and Sensitivity to Rejection (MSR) scales. *Current Psychology, 13,* 97-116.

Mehrabian, A., & Hines, M. (1978). A questionnaire measure of individual differences in dominance-submissiveness. *Educational and Psychological Measurement, 38,* 479-484.

Mehrabian, A., & Stefl, C.A. (1995). Basic temperament components of loneliness, shyness, and conformity. *Social Behavior and Personality, 23,* 253-264.

Mehta, P.H., Jones, A.C., & Josephs, R.A. (2008). The social endocrinology of dominance: Basal testosterone predicts cortisol changes and behavior following victory and defeat. *Journal of Personality and Social Psychology, 94,* 1087-1093.

Mehta, P.H., & Josephs, R.A. (2006). Testosterone change after losing predicts the decision to compete again. *Hormones and behavior, 50,* 684-692.

Mehta, P.H., & Josephs, R.A. (2010). Testosterone and cortisol jointly regulate dominance: Evidence for a dual-hormone hypothesis. *Hormones and Behavior, 58*, 898-906.

Mehta, P.H., Wuehrmann, E.V., & Josephs, R.A. (2009). When are low testosterone levels advantageous? The moderating role of individual verus intergroup competition. *Hormones and Behavior, 56,* 158-162.

Monden, Y., Koshiyama, K., Tanaka, H., Mizutani, S., Aono, T., Hamanaka, Y., …Matsumoto, K. (1972). Influence of major surgical stress on plasma testosterone, plasma LH, and urinary steroids. *European Journal of Endocrinology, 69,* 542-552.

Montoya, E.R., Terberg, D., Bos, P.A., & van Honk, J. (2012). Testosterone, cortisol, and serotonin as key regulators of social aggression: A review and theoretical perspective. *Motivation and Emotion, 36(1),* 65-73.

Mueller, A., Brocke, B., Fries, E., Lesch, K.P., & Kirschbaum, C. (2010). The role of the serotonin transporter polymorphism for the endocrine stress response in newborns. *Psychoneuroendocrinology, 35,* 289-296.

Murakami, F., Shimomura, T., Kotani, K., Ikawa, S., Nanba, E., & Adachi, K. (1999). Anxiety traits associated with a polymorphism in the serotonin transporter gene regulatory region in the Japanese. *Journal of Human Genetics, 44,* 15-17.

Nelissery, M., Feinn, R.S., Covault, J., Gelernter, J., Anton, R.F., Pettinati, H.,

…Kranzler, H.R. (2003). Alleles of a functional serotonin transporter promoter polymorphism are associated with major depression in alcoholics. *Alcoholism: Clinical and Experimental Research, 27,* 1402-1408.

Nettle, D. (2005). An evolutionary approach to the extraversion continuum. *Evolution and Human Behavior, 26,* 363-373.

Nettle, D. (2006). The evolution of personality variation in humans and other animals. *American Psychologist, 61,* 622-631.

Newman, M.L., Guinn Sellers, J., & Josephs, R.A. (2005). Testosterone, cognition, and social status. *Hormones and Behavior, 47,* 205-211.

Nisbett, R.E., & Wilson, T.C. (1977). Telling more than we know: Verbal reports on mental processes. *Psychological Review, 84,* 231-259.

Noskova, T., Pivac, N., Nedic, G., Kazantseva, A., Gaysina, D., Faskhutdinova, G., …Seler, D.M. (2008). Ethnic differences in the serotonin transporter polymorphism (5-HTTLPR) in several European populations. *Progress in Neuro-psychopharmacology and Biological Psychiatry, 32,* 1735-1739.

Oliveira, T., Gouveia, M.J., & Oliveria, R.F. (2009). Testosterone responsiveness to winning and losing experiences in female soccer players. *Psychoneuroendocrinology, 34,* 1056-1064.

Osinsky, R., Reuter, M., Kupper, Y., Schmitz, A., Kozyra, E., Alexander, N., & Hennig, J. (2008). Variation in the serotonin transporter gene modulates selective attention to threat. *Emotion, 8(4),* 584-588.

Ozer, D.J., & Benet-Martinez, V. (2006). Personality and the prediction of consequential outcomes. *Annual Review of Psychology, 57,* 401-421.

Penke, L., & Asendorpf, J.B. (2008). Beyond global sociosexual orientations: A more differentiated look at sociosexuality and its effects on courtship and romantic

relationships. *Journal of Personality and Social Psychology, 95(5),* 1113-1135.

Penke, L., & Denissen, J.J.A. (2008). Sex differences and lifestyle-dependent shifts in the attunement of self-esteem to self-perceived mate value: Hints to an adaptive mechanism. *Journal of Research in Personality, 42(4),* 1123-1129.

Penke, L., Denissen, J.J.A., & Miller, G.F. (2007). The evolutionary genetics of personality. *European Journal of Personality, 21,* 549-587.

Perez, M., Brown, J.S., Vrshek-Schallhorn, S., Johnson, F., & Joiner, Jr., T.E. (2006). Differentiation of obsessive-compulsive-, panic-, obsessive-compulsive personality-, and non-disordered individuals by variation in the promoter region of the serotonin transporter gene. *Anxiety Disorders, 20,* 794-806.

Perez-Edgar, K., Bar-Haim, Y., McDermott, J.M., Gorodetsky, E., Hodgkinson, C.A., Goldman, D., …Fox, N.A. (2010). Variations in the serotonin-transporter gene are associated with attention bias patterns to positive and negative emotion faces. *Biological Psychology, 83,* 269-271.

Plaistow, S.J., Johnstone, R.A., Colegrave, N., & Spencer, M. (2004). Evolution of alternative mating tactics: conditional versus mixed strategies. *Behavioral Ecology, 15,* 534-542.

Plomin, R., DeFries, J.C., McClearn, G.E., & Rutter, M. (2001). *Behavioral genetics* (4th ed.). New York: Freeman.

Pluess, M., Belsky, J., Way, B.M., & Taylor, S.E. (2010). 5-HTTLPR moderates effects of current life events on neuroticism: Differential susceptibility to environmental influences. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34,* 1070-1074.

Popma, A., Vermeiren, R., Geluk, C.A.M.L., Rinne, T., van den Brink, W., Knol, D.L., …Doeleijers, T.A.H. (2007). Cortisol moderates the relationship between

testosterone and aggression in delinquent male adolescents. *Biological Psychiatry, 61,* 405-411.

Power, T., Stewart, R., Ancelin, M., Jaussent, I., Malafosse, A., & Ritchie, K. (2010). 5-HTTLPR genotype, stressful life events and late-life depression: No evidence of interaction in French population. *Neurobiology of Aging, 31(5),* 886-887.

Pratto, F., Sidanius, J., Stallworth, L.M., & Malle, B.F. (1994). Social dominance orientation: A personality variable predicting social and political attitudes. *Journal of Personality and Social Psychology, 67,* 741-763.

Putnam, P., Hermans, E.J., & van Honk, J. (2004). Emotional stroop performance for masked angry faces: It's BAS, not BIS. *Emotion, 4(3),* 305-311.

Radloff, L.S. (1977). The CES-D scale: A self report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.

Regan, P.C. (1998a). Minimum mate selection standards as a function of perceived mate value, relationship context, and gender. *Journal of Psychology and Human Sexuality, 10(1),* 53-73.

Regan, P.C. (1998b). What if you can't get what you want? Willingness to compromise ideal mate selection standards as a function of sex, mate value, and relationship context. *Personality and Social Psychology Bulletin, 24(12),* 1294-1303.

Relyea, R.A. (2002). Costs of phenotypic plasticity. *American Naturalist, 159,* 272-282.

Roney, J.R., Lukaszewski, A.W., & Simmons, Z.L. (2007). Rapid endocrine responses of young men to social interactions with young women. *Hormones and Behavior, 52,* 326-333.

Roney, J.R., Mahler, S.V., & Maestripieri, D. (2003). Behavioral and hormonal responses of men to brief interactions with women. *Evolution and Human Behavior, 24,* 365-375.

Roney, J.R., Simmons, Z.L., & Lukaszewski, A.W. (2010). Androgen receptor gene sequence and basal cortisol concentrations predict men's hormonal responses to potential mates. *Proceedings of the Royal Society B, 277,* 57-63.

Rose, R.M., Bernstein, I.S., & Gordon, T.P. (1975). Consequences of social conflict on plasma testosterone levels in rhesus monkeys. *Psychosomatic Medicine, 37(1),* 50-61.

Rose, S., & Frieze, I.H. (1993). Young singles' scripts for a first date. *Gender and Society, 3(2),* 258-268.

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.

Rowe, D.C., Vazsonyi, A.T., & Figueredo, A.J. (1998). Mating effort in adolescence: A conditional or alternative strategy. *Personality and Individual Differences, 23,* 105-115.

Saad, G., & Vongas, J.G. (2009). The effect of conspicuous consumption on men's testosterone levels. *Organizational Behavior and Human Decision Processes, 110,* 80-92.

Salvador, A. (2011). Steroid hormones and some evolutionary-relevant social interactions. *Motivation and Emotion, 36(1),* 74-83.

Salvador, A., Suay, F., Gonzalez-Bono, E., & Serrano, M.A. (2003). Anticipatory cortisol, testosterone, and psychological responses to judo competition in young men. *Psychoneuroendocrinology, 28(3),* 364-375.

Schinka, J.A., Busch, R.M., & Keene-Robichaux, N. (2004). A meta-analysis of the association between the serotonin transporter gene polymorphism (5-HTTLPR) and trait anxiety. *Molecular Psychiatry, 9,* 197-202.

Schmidt, L.A., Fox, N.A., Rubin, K.H., Sternberg, E.M., Gold, P.W., Smith, C.C., &

Schulkin, J. (1997). Behavioral and neuroendocrine responses in shy children. *Developmental Psychology, 30(2),* 127-140.

Schmitt, D.P. (2005). Fundamentals of human mating strategies. In D.M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 258-291). Hoboken, NJ: John Wiley & Sons.

Schmitz, A., Hennig, J., Kuepper, Y., & Reuter, M. (2007). The association between neuroticism and the serotonin transporter polymorphism depends on structural differences between personality measures. *Personality and Individual Differences, 42,* 789-799.

Schultheiss, O.C., Wirth, M.M., Torges, C.M., Pang, J.S., Villacorta, M.A., & Welsh, K.M. (2005). Effects of implicit power motivation on men's and women's implicit learning and testosterone changes after social victory or defeat. *Journal of Personality and Social Psychology, 88(1),* 174-188.

Schulz, P., Walker, J.P., Peyrin, L., Soulier, V., Curtin, F., & Steimer, T. (1996). Lower sex hormones in men during anticipatory stress. *NeuroReport, 7,* 3101-3104.

Sellers, J.G., Mehl, M.R., & Josephs, R.A. (2007). Hormones and personality: Testosterone as a marker of individual differences. *Journal of Research in Personality, 41,* 136-138.

Segerstrom, S.C., & Miller, G.E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130(4),* 601-630.

Shackelford, T.K. (2006). Recycling, evolution and the structure of human personality. *Personality and Individual Differences, 41,* 1551-1556.

Shoal, G., Giancola, P., & Kirillova, G. (2003). Salivary cortisol, personality, and aggressive behavior in adolescent boys: A 5-year longitudinal study. *Journal of*

*the American Academy of Child and Adolescent Psychiatry, 42(9),* 1101-1107.

Simerly, R.B., Chang, C., Muramatsu, M., & Swanson, L.W. (1990). Distribution of androgen and estrogen receptor mRNA-containing cells in the rat brain: an in situ hybridization study. *Journal of Comparative Neurology, 294,* 76-95.

Singelis, T.M. (1994). The measurement of independent and interdependent self-construals. *Personality and Social Psychology Bulletin, 20,* 580-591.

Singh, D. (1993). Adaptive significance of female physical attractiveness: Role of waist-to-hip ratio. *Journal of Personality and Social Psychology, 65,* 293-307.

Smolewska, K.A., McCabe, S.B., & Woody, E.Z. (2006). A psychometric evaluation of the Highly Sensitive Person Scale: The components of sensory-processing sensitivity and their relation to BIS/BAS and "Big Five." *Personality and Individual Differences, 40(6),* 1269-1279.

Speilberger, C.D., Gorsuch, R.L., & Lushene, R.E. (1970). *Manual for the state-trait anxiety inventory*. Palo Alto, CA: Consulting Psychologists Press.

Stogdill, R.M. (1963). *Manual for the leadership behavior description questionnaire-xii*. Columbus, OH: Ohio State University Press. College of Administrative Science.

Surbey, M.K., & Brice, G.R. (2007). Enhancement of self-perceived mate value precedes a shift in men's preferred mating strategy. *Acta Pscyhologica Sinica, 39(3),* 513-522.

Surtees, P.G., Wainwright, N.W.J., Willis-Owen, S.A.G., Luben, R., Day, N.E., & Flint, J. (2006). Social adversity, the serotonin transporter (5-HTTLPR) polymorphism and major depressive disorder. *Biological Psychiatry, 59,* 224-229.

Symons, D. (1979). *The evolution of human sexuality*. New York: Oxford University Press.

Takahashi, T. (2004). Cortisol levels and time-discounting of monetary gain in humans.

*NeuroReport, 15(13),* 2145-2147.

Taylor, S.E., Way, B.M., Welch, W.T., Hilmert, C.J., Lehman, B.J., & Elsenberger, N.I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry, 60,* 671-676.

Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual. The role of genetics and adaptation. *Journal of Personality, 58,* 17-68.

Troisi, A. (2005). The concept of alternative strategies and its relevance to psychiatry and clinical psychology. *Neuroscience and Biobehavioral Review, 29,* 159-168.

Umekage, T., Tochigi, M., Marui, T., Kato, C., Hibino, H., Otani, T.,…Sasaki, T. (2003). Serotonin transporter-linked promoter region polymorphism and personality traits in a Japanese population. *Neuroscience Letters, 337,* 13-16.

van Honk, J., Harmon-Jones, E., Morgan, B.E., & Schutter, D.J.L.G. (2010). Socially explosive minds: The triple-imbalance hypothesis of reactive aggression. *Journal of Personality, 78(1),* 67-94.

van Honk, J., Peper, J.S., & Schutter, D.J.L.G. (2005). Testosterone reduces unconscious fear but not consciously experienced anxiety: Implications for disorders of fear and anxiety. *Biological Psychiatry, 58,* 218-225.

van Honk, J., Schutter, D.J.L.G., Hermans, E.J., & Putman, P. (2003). Low cortisol levels and the balance between punishment sensitivity and reward dependency. *NeuroReport, 14,* 1993-1996.

van Honk, J., Schutter, D.J.L.G., Hermans, E.J., Putman, P., Tuiten, A., & Koppeschaar, H. (2004). Testosterone shifts the balance between sensitivity for punishment and reward in healthy young women. *Psychoendocrinology, 29,* 937-943.

van Honk, J., Tuiten, A., Hermans, E., Putman, P., Koppeschaar, H., Thijssen, J., et al. (2001). A single administration of testosterone induces cardiac accelerative responses to angry faces in healthy young women. *Behavioral Neuroscience, 115,* 238-242.

van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., & Verbaten, R. (1998). Baseline salivary cortisol levels and preconscious selective attention for threat: A pilot study. *Psychoneuroendocrinology, 23,* 741-747.

van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., Thijssen, J., & de Haan, E. (1999). Correlations among salivary testosterone, mood, and selective attention to threat in humans. *Hormones and Behavior, 36,* 17-24.

van Wingen, G., Mattern, C., Verkes, R.J., Buitelaar, J., & Fernandez, G. (2010). Testosterone reduces amygdala-orbitofrontal cortex coupling. *Psychoneuroendocrinology, 35,* 105-113.

Verschoor, E., & Markus, R. (2011). Affective and neuroendocrine stress reactivity to an academic examination: Influence of the 5-HTTLPR genotype and trait neuroticism. *Biological Psychiatry, 87,* 439-449.

Verschoor, E., & Markus, R. (in press). Physiological and affective reactivity to a 35% CO2 inhalation challenge in individuals differing in the 5-HTTLPR genotype and trait neuroticism. *European Neuropsychopharmacology*.

Viau, V. (2002). Functional cross-talk between the hypothalamic-pituitary-gonadal and adrenal axes. *Journal of Neuroendocrinology, 14,* 506-513.

Vinberg, M., Mellerup, E., Andersen, P.K., Bennike, B., & Kessing, L.V. (2010). Variations in 5-HTTLPR: Relation to familiar risk of affective disorder, life events, neuroticism and cortisol. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34,* 86-91.

von Rueden, C., Gurven, M., & Kaplan, H. (2011). Why do men seek status? Fitness payoffs to dominance and prestige. *Proceedings of the Royal Society B, 278,* 2223-2232.

Vormfelde, S.V., Hoell, I., Tzvetkov, M., Jamrozinski, K., Sehrt, D., Brockmoller, J., & Leibing, E. (2006). Anxiety- and novelty seeking-related personality traits and serotonin transporter gene polymorphisms. *Journal of Psychiatric Research, 40,* 568-576.

Wagner, J.D., Flinn, M.V., & England, B.G. (2002). Hormonal responses to competition among male coalitions. *Evolution and Human Behavior, 23,* 437-442.

Wakefield, J.C. (2005). Biological function and dysfunction. In D.M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 878-902). Hoboken, NJ: John Wiley & Sons.

Way, B.M., & Taylor, S.E. (2010). The serotonin transporter promoter polymorphism is associated with cortisol response to psychosocial stress. *Biological Psychiatry, 67,* 487-492.

Wenegrat, B., Abrams, L., Castillo-Yee, E., & Romine, I.J. (1996). Social norm compliance as a signaling system. I. Studies of fitness-related attributions consequent on everyday norm violations. *Ethology and Sociobiology, 17,* 403-416.

Wenegrat, B., Castillo-Yee, E., & Abrams, L. (1996). Social norm compliance as a signaling system. II. Studies of fitness-related attributions consequent of a group norm violation. *Ethology and Sociobiology, 17,* 417-429.

Whisman, M.A., Richardson, E.D., & Smolen, A. (2011). Behavioral inhibition and triallelic genotyping of the serotonin transporter promoter (5-HTTLPR) polymorphism. *Journal of Research in Personality, 45,* 706-709.

Wilhelm, K., Mitchell, P.B., Niven, H., Finch, A., Wedgwood, L., Scimone, A., …Schofield, P.R. (2006). Life events, first depression onset and the serotonin transporter gene. *British Journal of Psychiatry, 188,* 210-215.

Wilson, D.S. (1998). Adaptive individual differences within single populations. *Philosophical Transactions: Biological Sciences, 353,* 199-205.

Wilson, T.C., & Nisbett, R.E. (1978). The accuracy of verbal reports about the effects of stimuli on evaluations and behavior. *Social Psychology, 41,* 118-131.

Wolf, M., van Doom, G.S., Leimar, O., & Weissing, F.J. (2007). Life-history trade-offs favour the evolution of animal personalities. *Nature, 447,* 581-585.

Yao, S., Luo, Y., Yang, J., Wang, X., & Zhu, X. (2009). Neuroticism as a vulnerability factor to depression symptoms in university students: A mulit-wave longitudinal study. *Chinese Journal of Clinical Psychology, 17,* 598-600.

Yu, Y., & Shi, J. (2009). Relationship between levels of testosterone and cortisol in saliva and aggressive behaviors in adolescents. *Biomedical and Environmental Sciences, 22,* 44-49.

Zalsman, G., Huang, Y., Oquendo, M.A., Burke, A.K., Hu, X., Brent, D.A., …Mann, J.J. (2006). Association of a triallelic serotonin transporter gene promoter region (5-HTTLPR) polymorphism with stressful life events and severity of depression. *American Journal of Psychiatry, 163,* 1588-1593.

Zyphur, M.J., Narayanan, J., Koh, G., & Koh, D. (2009). Testosterone-status mismatch lowers collective efficacy in groups: Evidence from a slope-as-predictor multilevel structural equation model. *Organizational Behavior and Human Decision Processes, 110,* 70-79.